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BICHAT.[1]

By William Sydney Thayer, M. D.,

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It is fitting that in 1902, the one hundredth anniversary of the death of Bichat, we should contemplate for a moment the career of one of the most remarkable men of modern times. Marie François Xavier Bichat was born in Thoirette in the province of Bresse, now Jura, on the 14th of November, 1771. His father was a physician and mayor of Poncin in Bugey. The early life of young Bichat appears to have been uneventful. It was the wish of the father that his son should follow in his footsteps, and whatever advantages one may gain from being brought up in a medical atmosphere the young man seems to have had. Bichat was brilliant from infancy and history shows that he was a striking exception to the common rule, in that this youthful precocity was in no way delusive. At school in Nantua and at the Seminary of Saint Yrénée in Lyon, he gained prizes and honors, and soon manifested a tendency toward mathematics and the natural sciences, especially physics and natural history. Beginning the study of anatomy at the hospital in Lyon under the direction of the celebrated Petit, he made good progress despite the fact that he appears at times to have allowed his exuberant energy and spirits to carry him into channels which deviated somewhat from those which ordinarily lead to a career in science. As Levacher[2] says, he was at this period more fitted to take a rapid bird's-eye view of all parts of the subject than to devote himself to the profound study of a single branch. But his memory and power of comprehension were remarkable, and early in his career he began to apply the accurate methods of thought and investigation which he had learned in mathematics and physics to the study of the structure and functions of man.

Returning home to pursue his surgical work with his father, he was soon drawn again to Lyon and to his mathematical studies, without, however, abandoning anatomy. But in 1793, the stormy days of the Revolution closed the doors of all institutions of learning at Lyon and drove Bichat forever from the scenes of his boyhood. After a short sojourn in Bourg he went

[1] Read before the Johns Hopkins Historical Society, December 8, 1902.

[2] Levacher de la Feutrie, Éloge de Bichat, Mém. Soc. d'émulat, Par., 1803, v, pp. xxvii-lxiv.

to Paris to continue his studies which were to fit him for the position of surgeon in the army. Arriving in Paris without friends or letters of introduction he devoted himself especially to the clinic of the celebrated surgeon Desault, to whose attention he was brought soon after his arrival, through circumstances happily related by Buisson.[3] "It was an established custom in the school of Desault that certain chosen pupils should undertake to collect in turn the public lesson and prepare an extract. This extract was read after the lesson of the following day; and these exercises, presided over by the associate surgeon, had the double advantage of bringing a second time before the pupils the useful precepts which they should absorb, and of making up for the sufficiently common

inattention of the masses during the first lesson. One day when Desault had spoken for a long time on a fracture of the clavicle, and had demonstrated the utility of his bandage, applying it at the same time to a patient, the pupil whose duty it was to collect these details happened to be absent. Bichat offered to take his place. The reading of his extract caused a real sensation. The purity of his style, the precision and clearness of his ideas, the scrupulous exactness of his résumé were characteristic rather of the professor than the pupil. He was heard with extraordinary attention and left showered with praise and repeated applause." When informed of the incident by his associate Manoury, Desault straightway sought the acquaintance of this promising disciple, in whom he soon recognized a man of genius. The master not only opened his house to the pupil, but practically adopted him as a son, and throughout the remaining years of his life Bichat was associated with all the work of his teacher. This was the turning point in his life. His association with Desault opened to him the opportunity for a scientific career to which his whole energy was afterwards devoted.

In 1795 Desault died. Though a great sorrow to Bichat, the death of his master and benefactor in no way interrupted his career. He continued to live with Desault's widow who thenceforth regarded him as a son. From the death of Desault, Bichat gave himself up to a career of unremitting activity such as has rarely been equaled. In the very year of Desault's death he published his journal with an historical notice of his life and letters, and later edited his surgical works. But his main energies were devoted to the study of anatomy. The knowledge of anatomy he found in a condition which may justly be called chaotic. As one biographer has said,[4] "The general anatomy of man was unknown." Much that was taught consisted of a mass of hypothetical or dogmatic statements which had been handed down from master to pupil for ages. In the words of Husson,[5] "Up to that time bristling with scholastic minutiæ, anatomy repelled too often by its dryness the young who were destined to the study of the healing art. We cannot even to-day" [the year of Bichat's death] "recall without a sensation of pain all those multiple divisions, those fatiguing descriptions, that conventional and often incomprehensible language which constituted then the science of anatomy. Bichat was the first to leave the common path; he presented anatomy in a new point of view; studied the general organization of man in the simple tissues of which he is composed, divided the living economy into various systems, and by accumulating facts, by bringing observation to bear on experience, he broadened the limits of science and built for himself a monument which brings him lasting renown." Bichat's great work consisted in the introduction into anatomy and physiology of methods of accurate, systematic observation and experiment, methods similar to those which distinguished the later clinical schools of Laennec, Louis, and the physiological studies of Claude Bernard. "Anatomy," said he one day to his colleagues, "is not as they teach it to us, and physiology is a science to be made over again."[6] Bichat devoted himself literally day and night to his studies, and unmoved by the stirring and distracting incidents of this turbulent period, lived among his cadavers, writing the protocols of his observations and experiments in the small hours of the night. He soon gathered about him enthusiastic pupils and friends to whom in 1797 he gave his first course in anatomy. "From this moment," says Levacher,[7] "one must measure his success by his productions, and his years by his successes."

[3] Buisson: De la division la plus naturelle des phénomènes physiologiques considérée chez l'homme, avec un précis historique sur M. F. X. Bichat. 344 pp., 8°, Paris, Brosson, an. x (1802), p. 326.

[4] Knox: Lancet, Lond., 1854, ii, 393.

[5] Notice historique sur la vie et les travaux de Marie-Fr.-Xav.-Bichat, in Traité des membranes &c. par X. Bichat. Nouvelle édition augmentée d'une notice &c. par M. Husson., Par., 8°, Ann. xi, 1802.

[6] Pariset: Discours à l'inauguration de la statue de Bichat à Bourg. Gaz. méd. de Par., 1843, 2 s., xi, Ann. xiv, 566.

[7] Loc. cit.

He was one of the founders of the Société médicale d'émulation, in the proceedings of which his earlier works were published:

Description d'un nouveau trépan (vol. ii des Mémoires de la Soc. méd. d'émulation).

Mémoire sur la fracture de l'extrémité scapulaire de la clavicule (Ibid.).

Description d'un procédé nouveau pour la ligature des polypes (Ibid.).

Mémoire sur la membrane synoviale des articulations (Ibid.).

In this latter publication he introduces his ideas concerning the distinction of tissues which were afterwards elaborated in his general anatomy. Synovial membranes are described for the first time.

Dissertation sur les membranes et sur leurs rapports généraux d'organization" (Ibid.).

Next there appeared: *Mémoire sur les rapports qui existent entre les organes à formes symetriques et ceux à forme irregulière* (Ibid.).

Here he introduces his theory of the two lives, the animal and organic.

In the same year, when barely twenty-nine years of age, he was appointed adjunct physician to the Hotel Dieu.

Finally, in 1800, he published his first great work, *"Traité des membranes en général et de diverses membranes en particulier," Par., 8°, 1800,* an admirable example of accurate, systematic anatomical description. Here he recognized the fact that not only the organism as a whole, but individual organs are composed of various tissues which may be distinguished one from another and which have notable individual characteristics. In his own words: "Chemistry has its simple bodies which by the diverse combinations to which they are susceptible, form compound bodies . . . In like manner anatomy has its simple tissues which by their combinations . . . form the organs."[8]

During the same year he published what is perhaps his most celebrated work, "*Recherches physiologiques sur la vie et la mort.*"

This work consists of two parts: the first theoretical, in which he distinguishes the animal life from organic life; the second, experimental, in which he endeavors to determine the rôle of the brain, the heart and the lungs in producing death. The book contains a mass of interesting physiological observations and theories, many of which are classical. Such, for instance are his observations on the action of red blood on the life of the brain, the action of venous blood in various functions, the functional independence of the brain and the heart. His theories, though ingenious, have in many instances suffered modifications with the lapse of years, but his observations and methods of research are models for all time.

Bichat's position in the Hotel Dieu gave him increased opportunities for the study of disease in the living and for the comparison of clinical and anatomical observations. Of these opportunities he made the most, seeking every chance to add to his experience by acting as substitute for his colleagues. In one winter he made over six hundred autopsies.

In 1801 appeared his *"Anatomie générale appliquée à la physiologie et à la médecine,"* a work memorable not only for its anatomical observations but for the remarkable applications which Bichat makes of these observations to physiology and pathology. "Pathological anatomy," says Cérise," "which

was but a collection of isolated facts, is here raised to the rank of a science. . . . Medical genius has never at a single bound raised itself to so great a height." In the preface to this treatise he speaks of the methods of study which have led him to the results set forth in the work. How modern are his words! "Experiments on living animals, tests with various reagents on organized tissues, dissections, necropsies, observation of man in health and disease, these are the sources from which I have drawn; they are those of nature. Nor have I neglected those of the authors, especially of those for whom the science

[8] Anatomie générale &c., p. lxxix.

[9] Cérise: Œuvres du Docteur, Par., 1872, vol. ii, p. 403.

of the animal economy has been a science of facts and experience." Nature was indeed his text-book, and on one occasion he is reported to have said, "If I have made such rapid headway, it is because I have read little." [10]

The relations of pathological anatomy to clinical medicine have rarely been better expressed than in the following words: "We are, it seems to me, at a point where pathological anatomy must take a new flight. It is not alone the science of those changes which primarily or secondarily develop gradually in the course of chronic disease; it includes the examination of every alteration to which our parts are subject at whatsoever period of the disease. . . . How petty are the reasonings of a multitude of physicians great in the eye of the public, when investigated not by the light of their own writings, but in the cadaver! Medicine has been for a long time excluded from the exact sciences; it will have a right to be associated with them at least as regards the diagnosis of disease when one shall have combined everywhere with rigorous clinical observation the examination of the alterations suffered by our organs. . . . Of what value is clinical observation if one is ignorant of the seat of the evil? You might take notes at the bedside of the sick for twenty years from morning to night on affections of the heart, of the lung, of the abdominal viscera, &c., and there will be but confusion in the symptoms, which resting upon no certain base, will of necessity bring before you an incoherent sequence of phenomena. Open a few cadavers and that obscurity which clinical observation alone could never have dissipated will vanish in a moment from before your eyes." [11]

Realizing early the traditional, blind, therapeutic empiricism which then prevailed, Bichat became deeply interested in the physiological action of drugs and made many careful, systematic pharmacological experiments on animals. His power for work was little short of marvelous. He began a treatise upon descriptive and pathological anatomy, working all day and writing much of the night, but with such extraordinary rapidity, accuracy and clearness, that his pages are said to have gone unread and uncorrected from his pen to the printer. Young, attractive and spirited, the few moments which he snatched for the more ardent pleasures of life, came not from his hours of work, but from those which should have been devoted to rest and recuperation—and the end was the old familiar one. One day an attack of hæmoptysis, the moments of discouragement soon forgotten, the old manner of life renewed, several repetitions of the accident, frequent "gastric disturbances," and finally, after a hot July day spent among decomposing bodies in an atmosphere so foul that it had driven all his associates from the laboratory, an attack of syncope, a fall, followed shortly by an "ataxic fever" which proved fatal on the 14th day, the 22nd of July, 1802. He died in the arms of his master's widow, to whom he had been for seven years a devoted son.

[10] The Practitioner, Lond., 1896, lvi, 280.

[11] Bichat: Anatomie générale, &c. Nouvelle édit., Paris, 1812, Brosson et Gabon, t. I, p. xcviii.

"He was," says Larrey, [12] "but thirty years old, but he was already the greatest physiologist of his century, as he must have been the greatest physician had he but lived twenty years more."

Bichat seems to have possessed, in addition to his genius, a character in many ways remarkable. With all his powers and his restless energy, he was a modest, affectionate and singularly lovable man, incapable of jealousy or resentment and devoted to his friends. "If," says Pariset, [13] "we may believe Fénelon, few men have the strength to support the talents which they have received from heaven. I venture to assert that Bichat belonged to this small number of favored men." Roux, [14] a companion and student, who for several years held most intimate relations with Bichat, speaks feelingly of his remarkable

and genuine modesty. But now and then some slight act would reveal to his more intimate associates the consciousness of his own strength, and once, in a tête-a-tête with Roux he said, apropos of his own career: "J'irai loin, je crois."

He died without leaving the wherewithal to provide for his funeral, but he was piously cared for by his friends, while all the professors of the faculty and 600 students followed his remains to the grave.

His death caused profound emotion throughout the medical profession. Corvisart wrote to Napoleon, then first consul: [15]

[12] Larrey: Discours prononcé à l'inauguration de la statue de Bichat, 8°, Paris, 1843; also Gaz. méd. de Par., 1843, xiv, 2 s., xi, 569.

[13] Loc. cit.

[14] Gaz. méd. de Par., 1845, 2 s., xiii, 763.

[15] Larrey: Inauguration de la statue de Bichat le 16 juillet, 1857, à la Faculté de Médecine de Paris. Discours de Larrey au nom de la Société médicale d'émulation.

"Bichat has just died at the age of 30; he has fallen upon a field of battle which, also, calls for courage, and which counts many a victim; he has broadened the science of medicine; no one at his age has done so many things and done them so well." And Napoleon, wishing to honor both Bichat and his master, wrote to the Minister of the Interior: "I beg that you will have placed in the Hotel Dieu a marble dedicated to the memory of Citizens Desault and Bichat which shall attest the gratitude of their contemporaries for the service which they have rendered, one to French surgery, of which he is the restorer, the other to medicine, which he has enriched by many useful works. Bichat would have broadened the domain of this science, so important and so dear to humanity, if pitiless death had not struck him down at the age of 30." The monumental stone upon the wall of the peristyle of the Hotel Dieu has for an inscription an extract from this memorable letter.

Since this time the memory of Bichat has not faded. In 1833 the Société d'émulation de Jura erected a commemorative stone by the house in which he was born in Thoirette. In 1837, David, charged with the duty of designing the frieze on the façade of the Pantheon upon which are inscribed the fine words, "Aux grands hommes la partie reconnaissante," represents Bichat dying, his head crowned with laurels. In one hand he holds a pen, and in the other the manuscript of his work, "Sur la vie et la mort."

In 1839 a monument was erected in honor of the memory of Bichat at Son-le-Saulnier, *chef-lieu* of the department of Jura, a column surmounted by a bronze bust by Huguenin.

In 1843 a fine memorial was dedicated at Bourg en Bresse. The statue by David (d'Angers) represents Bichat in an attitude of meditation, his hand seeking the impulse of the heart of a child who stands by his side—at his feet a partly dissected body and a lamp, symbolizing the light which his genius had cast upon the obscurities of life and death.

In 1857 the statue, also by David, which stands in the quadrangle of the school of medicine at Paris was unveiled.

In 1844 the city at last granted a fitting burial place for Bichat at Père-La-chaise, and on the 16th of November, 1845, forty-three years after his death, his remains were solemnly exhumed before a committee of the Medical Congress of France and carried to Notre Dame, where obsequies were held; thousands marched in the funeral procession.

And again last summer the Société Française d'histoire de la médecine celebrated the centennial anniversary of his death by a visit to his tomb, the placing of an inscription upon the house in which he died, and literary exercises in which addresses were made by a number of distinguished members of the profession. A medal has been struck in honor of the occasion.

Looking back upon the life of this truly great man one cannot but feel the inspiration of it all. And though reason remind us that 'tis a career rather to admire than to emulate, yet one must be stirred by the fine words of Levacher,[16] addressed to the members of the society which owed so much to his influence and labors:

"Let Bichat be at the same time the guide and the model. He has shown what one could do in but a little while. What an example for you young men who are pursuing the same career. You are witnesses of the regret which he carries with him; of the tears which he has caused to flow, and of his triumphs; take him for an example. Be as he was, active and laborious, patient and zealous, and if you need to sustain yourselves in your work pronounce the name of Bichat. Remember above all that time adds nothing to glory and that with genius and work thirty years of life suffice to render one's name immortal."

[16] Mem. Soc. méd. d'emulat., Par., 1803, v, pp. xiv-xxvii.

SOME EARLY AUTOPSIES IN THE UNITED STATES.*

BY WALTER R. STEINER, M. D.,

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The date of the first autopsy performed in the United States is unknown. Toner[1] from two researches decided that the one made by Johannes Kerfbyle, a Dutch physician and a Leyden graduate, on Governor Slaughter, of New York, in 1691, was the first recorded. Packard[2] in his entertaining book on the history of medicine in the United States describes one done in 1674 as the earliest. He also relates four others of a little later period. There are, however, a number on record a good many years previous to these. For the references to all of them save the two performed in Maryland I am indebted to Dr. Hoadly's article.[3]

In September, 1639, Winthrop[4] tells us in his history of New England that Marmaduke Percy, of Salem, was arraigned for the death of his apprentice boy. "This boy was ill-disposed, and his master gave him unreasonable correction and used him ill in his diet. After, the boy gate a

* Read before the Johns Hopkins Hospital Historical Club, October 13, 1902.

[1] Toner, Contributions to the Annals of Medical Progress, Washington, 1874, p. 57.

[2] Packard, The History of Medicine in the United States, Philadelphia, 1901, pp. 62-63.

[3] Hoadly, Some Early Post-Mortem Examinations in New England. Proc. Conn. Med. Soc., Bridgeport, 1892, pp. 207-217.

[4] Winthrop, The History of New England, Boston, Edition of 1853, i, p. 384.

bruise on his head, so as there appeared a fracture in his skull, being dissected after his death."

The Maryland Archives contain an interesting account of a post-mortem examination, done in 1643, on an Indian lad who was killed by his master, John Dandy.[5] A jury composed of twelve men formed the inquest. Their foreman, George Binx, was a licentiate in physic and probably performed the autopsy. There was, however, another doctor on the jury, Robert Ellyson, a barber chirurgeon. The report reads as follows:

"We find that this Indian (named Edward) came by his death by a bullett shot by John Dandy which bullett entered the epigastrium near the navell on the right side, obliquely descending & piercing the gutts, glancing on the last vertebra of the back, and was lodged in the side of Ano.

foreman

George Binx."

Fourteen years later Mr. Richard Maddokes and Mr. Emperor Smith, chirurgeons, view the body of Henry Gouge, who was thought to have been killed by his master, the above John Dandy. The eleven men who formed the inquest report "that we can See nor find nothing about the Said head, but only two places of the Skin and flesh broke on the right Side of the head and the Scull perfect and sound, and not anything doth or can appear to us to be any cause of the Death of the said Gouge." They also endeavored to search the body, but could not possibly do it, it being "so noysome" to them all.[6]

Referring to the loss of Rev. Mr. Danforth's three children, in an epidemic in 1659, Cotton Mather[7] says that "the (until then unknown) malady of 'bladders in the wind-pipe' invaded and removed many children; by opening of one of them the malady and remedy (too late for very many) were discovered." Diphtheria and tracheotomy are probably here implied.

Elsewhere in his Magnalia, Mather[8] makes the following note: "As for Mr. Stone if it were *metaphorically* true (what they *proverbially* said) of Beza, that 'he had no gall,' the physicians that opened him after his death found it *literally* true of this worthy man." This autopsy on Rev. Mr. Stone, who was assistant pastor of the first church of Christ in Hartford, was probably performed by Dr. Bryan Rossiter, of Guilford, Conn., in July, 1663. Rossiter had previously prescribed for Stone, and had been paid ten pounds for it by the town of Hartford.[9]

"We do not know where Dr. Rossiter obtained his medical education, but Dr. Hoadly thinks it was in England. He came to this country, in 1630, with his father, and was made a freeman a year later of Dorchester, Mass. In 1639 he moved to Windsor, Conn., became town clerk there, and was admitted to practice in the State of Connecticut by the General Court, 'being first tried and approved by Mr. Hooker, Mr. Stone, and old Mr. Smith of Wethersfield, in the face of the said Court.'[10] He migrated later to Guilford, and was continually in hot water with the people of the town about the union of New Haven Colony with Connecticut. He finally moved to Killingworth (now Clinton), but soon returned and died in Guilford, in 1672. "His practice was very large and he was frequently called to see cases in all parts of the State."

In the following extract from a letter to his daughter and her husband, on September 24, 1669, we get some idea of his busy practice: "We have had a sore visitation again by sickness and mortality here in Guilford this summer, as the last. Our graves are multiplied and fresh earth heaps are increased. Coffins again and again have been carried out of my doors. I have taken up a lot amongst the tombs in the midst of them."[12] During this "visitation" he lost his wife, his daughter and a grandchild.

For some reason or other he incurred the enmity of Gov. Leete, of Guilford, and whenever medical services were required in Leete's family Gov. Winthrop, that worthy colonial physician and statesman, was appealed to for aid. In 1658, Leete[13] details to Winthrop an eye trouble which affected his son, Peregrine, aged nine weeks, and in a later letter[14] asks Winthrop's directions concerning cordial powder the latter furnished Leete's wife by John Crane, for Graciana their daughter. It seems that no information accompanied the powder about the taking of it. Leete says: "Truly one of the most needfull directions is how to make her willing & apt to take it; for though it seemes very pleasant of itselfe, yet is she grown so marveilous aukward & averse from takeing it in beer. Wherefore I would entreat you to prescribe to us the varyety of wayes in which it may be given soe effectually; wee doubt els it may doe much lesse good, being given by force only." On another occasion he writes in a foot-note,[15] "my wife entreats some more of your physick, although shee feareth it to have very contrary operations in Mr. Rossiters stomach"[15]—a suggestion that professional jealousy existed in those days.

Rossiter's fame as a physician called him also to Hartford, in 1662, to ascertain by an autopsy whether the child of John Kelly was bewitched. The child was a girl, aged eight years, who "was taken in the night following Sunday, March 23, 1661-2, with a violent attack of something like bronchopneumonia. In her delirium she cried out against Goody Ayres as choking her and afflicting her, and the last words the child

[5] Provincial Court, i, pp. 254-255 and 260.

[6] Provincial Court, ii, pp. 524-525. For a fuller account of these two autopsies see my paper entitled, A Contribution to the History of Medicine in the Province of Maryland. Johns Hopkins Hospital Bulletin, 1902, xiii, pp. 192-198.

[7] Mather, Magnalia, Hartford, Edition of 1853, i, p. 437.

[8] Mather, Magnalia, Hartford, Edition of 1853, ii, p. 64.

[9] Conn. Hist. Soc. Coll., Hartford, 1897, vi, p. 108.

[10] Stiles, Ancient Windsor, Hartford, 1891, i, p. 453. The above quotation is taken from a letter Rossiter wrote to Gov. John Winthrop, Jr., in 1669. It is given in full in the Bulletin of the N. Y. Public Library, 1899, iii, pp. 402-404.

[11] Steiner, History of Guilford, Conn., Baltimore, 1897, pp. 477-478.

[12] Smyth, Dr. Bryan Rossiter of Guilford, Conn., and his descendants. N. E. Hist. and Gen. Reg., Boston, 1901, lv, pp. 149-154.

[13] Mass. Hist. Coll., Boston, 1865. vii, 4 Series, p. 539.

[14] Mass. Hist. Coll., Boston, 1865, vii, 4 Series, p. 540.

[15] Mass. Hist. Coll., Boston, 1865, vii, 4 Series, p. 548.

spoke were to that effect."[16] Following the superstition of those times, both her parents and the town's people thought that her death was due to some preternatural cause. The town accordingly summoned a jury of six men to inquire of the cause or manner of her death. They report on March 31, 1662:

"We, whose names are underwritten, were called forth and desired to take notice of the dead child of John Kelley, do hereby testify that we saw as followeth: The child was brought forth and laid upon a form by Goodwife Rescoe and Goodwife Whaples, and the face of it being uncovered, Goodwife Ayres was desired by John Kelly to come up to it and to handle it. The child having purged a little at the mouth, Goodwife Ayres wiped the corner of the child's mouth with a cloth, and then she was desired to turn up the sleeve of the arm, and she did endeavor to do it, but the sleeve being somewhat strait she could not well do it. Then John Kelly himself ripped up both the sleeves of the arms, and upon the backside of both arms, from the elbow to the top of the shoulders were black and blue, as if they had been bruised or beaten. After this, the child was turned over upon the right side and so upon the belly, and then there came such a scent from the corpse as that it caused some to depart the room, as Gregory Wolterton and George Grave. Then the child being turned again and put into the coffin, John Kelly desired them to come into the room again to see the child's face, and then we saw upon the right cheek of the child's face a reddish tawny great spot, which covered a great part of the cheek, it being on the side next to Goodwife Ayres where she stood.

This spot or blotch was not seen before the child was turned, and the arms of the child did appear to be very limber in the handling of them.

Gregory Wolterton, The Mark of Thos. Catlinge,
Thomas Bull. Nath. Willett.
Joseph Nash, George Grave."

On the same day, that is five days after the child's death, Dr. Rossiter opened the body at the grave, described the appearance of the organs, noted the absence of rigor mortis, and mistook the signs of beginning decomposition for something supernatural, in the following protocol:

"All these particulars underwritten I judge preternatural.

Upon the opening of John Kelley's child at the grave I observed:

1. The whole body, the musculous parts, nerves and joints were all pliable, without any stiffness or contraction, the gullet only excepted. Experience of dead bodies renders such symptoms unusual.

2. From the costal ribs to the bottom of the belly in the whole latitude of the womb, both the scarf skin and the whole skin with the enveloping or covering flesh had a deep blue tincture, when the inward part thereof was fresh, and the bowels under it in true order, without any discoverable pecancy to cause such an effect or symptom.

3. No quantity or appearance of blood was in either venter or cavity, as belly or breast, but in the throat only at the very swallow, where was a large quantity as that part could well contain, both flesh and fluid, no way congealed or clodded, as it comes from a vein opened, that I stroke it out with my finger as water.

4. There was the appearance of pure fresh blood in the backside of the arm, affecting the skin as blood itself without bruising or congealing.

5. The bladder or gall was all broken and curded, without any tincture in the adjacent parts.

6. The gullet or swallow was contracted, like a hard fish bone, that hardly a large pease could be forced through.

Br: Rossiter."

Fortified by these findings John Kelly and Bethia, his wife, testify in open court on May 13, 1662, as to the alleged persecutions of their child by Goody Ayres, according to the child's testimony. They state that the child after eating some hot broth with the wife of William Ayres, against their wishes, complained of pain at her stomach. Her father gave her some angelica root which yielded her "present ease," but the relief was only temporary as some time later she died. Fearing then an indictment Goody Ayres fled suddenly with her husband, leaving their son, aged eight, behind them as well as all their possessions. We know nothing of their subsequent history.

At a General Assembly held at Hartford, March 11, 1662-63, the court allowed "unto Mr. Rossiter twenty pounds in reference to openinge Kellies child and his paynes to visit the Dep-Governo[r] and his paynes in visiting and administering to Mr. Talcot."[17]

[16] Hoadly, op. cit. I have been unable to consult the original papers. Dr. Hoadly says some of the material was obtained from Mr. John Carter Brown, of Providence, R. I. The Librarian, Mr. Winship, of the John Carter Brown Library, was unable to find any of the manuscripts for me.

[17] Conn. Col. Records, Hartford, 1853, i, p. 396.

THE JOHNS HOPKINS HOSPITAL BULLETIN.

The Hospital Bulletin contains details of hospital and dispensary practice, abstracts of papers read, and other proceedings of the Medical Society of the Hospital, reports of lectures, and other matters of general interest in connection with the work of the Hospital. It is issued monthly.

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A CASE OF NEURO-FIBROMATOSIS (VON RECKLINGHAUSEN'S DISEASE) WITH PARALYSIS AND MUSCULAR ATROPHY OF ARMS AND LEGS.[1]

By H. M. Thomas, M. D.,

Neurologist to the Johns Hopkins Hospital.

Dr. Osler has asked me to demonstrate this patient to the Society, on account of the very great interest of the nervous symptoms which she shows.

The patient is a woman from West Virginia, Cyrena W., æt. 51, single; admitted Oct. 17, 1902, to Dr. Osler's clinic; Med. No. 14,942. Complains of pain and stiffness all over.

The family history is unimportant. She has never heard of a case similar to hers occurring in the family.

She is the eldest of nine children. Has been always rather sickly. Went very little to school and has worked hard on a farm all her life. She had scarlet fever in infancy; diphtheria and measles when 27; "grippe" six years ago. Is subject to occasional headaches. Menstruated first at 16; was fairly regular from then up to 48, when menopause occurred.

Tumors of the skin have been present since early girlhood, but she paid little attention to them and can give little definite information about them. She has, however, noticed a great increase in the last four or five years and she has consulted a physician about them. Those on the face appeared last. She has noticed dark bluish spots on the body for many years.

About five years ago she began to suffer from a burning, stinging pain in her left ankle. This was not constant, but recurred from time to time and was so intense that at first she thought her skirts were on fire. The pains gradually affected more and more of the leg, extending up to the hip and back, the right leg being affected after the left. This pain was at first very severe; each attack would come on very suddenly and last only a second or two. She describes them very much as a tabetic describes lightning pains. For one or two years there has been some pain of a similar character in her arms and shoulders. There are no pains in the face except those which the patient refers to defective teeth.

With the onset of the pains, the patient began to have some difficulty in walking. Her brother first noticed that she limped a little, dragging one leg. The muscular weakness increased gradually and two years ago she had a good deal of difficulty in getting about, and for the last year has been entirely unable to walk. During this year she has spent her time between her bed and her chair, and up to a few weeks ago was able to get from bed to chair unassisted. For about four months her legs have been contracted at the knees and hips. For a little less than a year she has noticed some weakness in her arms, commencing in the right. This has gradually increased, and for about five months she has been unable to knit or sew, and has had difficulty in dressing herself. She has not dressed her hair for several months, but can still feed herself.

From the very first she has noticed muscular twitchings and from time to time she has had some singing in the ear.

She made no complaint about her bladder before admission, but since then has often had to be catheterized, although she is able, at times, to voluntarily pass a little urine.

For the last two or three months she has noticed some difficulty in swallowing—chokes easily. There has been no regurgitation through the nose. For about the same length of time she has noticed that her voice has become thick and weak. She complains of no trouble with her eyes or ears except slight tinnitus.

As you see, the patient is a poorly nourished woman, with a careworn expression. Scattered irregularly over the face, trunk, arms and legs there are numberless little tumors. These vary in size from the little ones, about as large as a pin-head, to those as large as a good-sized cherry, and some few even larger. (See Plate XIII.) Some of these tumors are definitely pedunculated and others are sessile. There are numerous pigmented areas scattered over the body and in several spots the skin has a bluish look, which is apparently due to the coming through of the nodular tumors. Some of the larger tumors feel lobulated and nodular, but most of the masses have a homogeneous consistency. Two of the tumors were excised for histological examination and show the ordinary structure of neurofibromata.

In so far as the skin manifestations go, the patient presents a typical picture of cutaneous neuro-fibromatosis. No definite tumors on the nerve-trunks have been made out, nor have we felt any definite plexiform growths, although the subcutaneous fat below the popliteal space has a curious cord-like feel.

Examination of the nervous system, however, reveals a very remarkable condition, and it is in this respect that I particularly wish to call your attention to the case. The patient is apathetic and somewhat dull. She speaks in a thick, muffled voice, suggesting some weakness of the laryngeal muscles. Her answers seem quite unreliable, especially in regard to dates. I could find no definite abnormality of any of the cranial nerves. There are no changes in the retina or the optic nerve. No definite disturbances of hearing could be made out, although the patient does complain from time to time of subjective auditory sensations. The muscles of the pharynx appear to act well; the tongue is protruded straight, but is slightly tremulous.

The muscles of the neck are strong but all the muscles moving the arms are more or less weak; there is no absolute loss of power in any of these muscles. The muscles of the arms are generally atrophied, but particularly so in the intrinsic muscles of the hand. Fibrillary tremors are noticed in the muscles of both arms, and I think can be seen by those who

[1] Presented to The Johns Hopkins Medical Society, November 17, 1902.

are near the patient. The deep reflexes are present in the arms and fairly active.

As you see, the patient lies on her back with her legs flexed at the hip and at the knee, and the ankle joints extended. There is not much limitation in the hip-joint to passive movements, although there is some contraction of the flexor muscles, but the knees cannot be fully extended and there is apparently some thickening of the knee-joints. One gets a sense of creaking when the knee-joints are moved. There is no limitation of movement of the ankle-joints. The legs are both almost completely paralyzed; no movements of the hip, knee or ankle-joint are present in either leg. The patient can flex slightly the toes of the right foot and in the left foot there is a slight flexion of the little toe. The legs are thin, showing a general atrophic condition of the muscles. Fibrillary tremors in the legs have not been noticed. The knee-kicks are absent and there is no reflex from the tendo-Achillis. Plantar irritation causes no response.

We have been unable to determine any noticeable loss of sensation throughout the body. Patient appreciates touch, pain and temperature as acutely as is normal. This is true for the skin in general, but over some of the nodules there is a distinct loss in the perception of pain; a prick of the pin is not felt as acutely as it is on the rest of the skin.

The facial nerves and muscles react normally to the electrical currents, as, in general, do the nerves and muscles of the arms, although there is, perhaps, some decrease in the excitability of the muscles of the right hand. In the legs there is a profound decrease in electrical excitability of all the nerves and muscles. The only nerves that can be stimulated are the internal popliteal, and to do this it requires currents of very great strength. The resulting muscular movement is a slight flexion of the ankle and of the toes. The muscles on the anterior aspect of the legs do not respond to any strength of galvanic current that I am willing to use. There is a slow response in the calf muscles when directly stimulated by a strong galvanic current.

The general physical examination has revealed no particular abnormality in her heart, lungs or other internal viscera. Her urine is practically normal. Blood: 70 per cent and 83 per cent of hæmoglobin; red blood corpuscles over five million, the leucocytes between seven and eight thousand per cm.

To recapitulate, the essential features of this case are: A single woman, 51 years old, with good heredity and nothing in her personal history of importance. Tumors of the skin have been present from her earliest childhood, but have increased markedly in the later years. For five years she has suffered from sharp, burning, stinging pains in her feet and legs. Weakness of the legs made its appearance with the pains and has gradually increased. For two years she has had great difficulty in getting about, and for the last year she has been unable to walk. Her arms have also become weak. Muscular twitchings have been noticed since the beginning of the weakness. Some weakness of the bladder.

You have seen a woman, covered with innumerable skin tumors, splotches of pigmentation and here and there bluish areas of the skin. She is apathetic and dull, speaks in a thick, muffled voice. We have made out no definite loss of function in any of the cerebral nerves. The muscles of the neck are strong, the arms are generally weak and somewhat atrophied, particularly so in the muscles of the hand. Fibrillary tremors are present in the arms. Deep reflexes in the arms are active. There is particularly complete paralysis of the muscles of both legs, the only voluntary movements being in the toes. Some flexor contracture in the hips and knees; none in ankles. The legs are atrophied; no fibrillary tremors are seen in them. The deep reflexes are lost; no plantar reflex. There is a very great decrease in electrical irritability in the nerves and muscles of legs, indeed in most of them it is abolished. There is some slight decrease in the electrical excitability in the forearm and hand. The facial muscles act normally. No objective sensory disturbance can be determined except over some of the skin tumors, where there is a dulling in the perception of pain.

The diagnosis of the skin condition presents no difficulty; the case is a very typical example of cutaneous neuro-fibromata—the so-called *fibroma molluscum*. These tumors, as von Recklinghausen has shown, are, for the most part, outgrowths of the connective tissue sheaths of the cutaneous nerves, and are often associated with similar outgrowths from other nerves, forming a condition generally called *diffuse or generalized neuro-fibromatosis*, or *von Recklinghausen's disease*. The classification of neuromata in general is in a somewhat chaotic state, and I have had copied the table which Alexis Thomson has suggested in his excellent monograph on neuroma and neuro-fibromatosis, published in Edinburgh in 1900.

The remarkable feature of this case is the association of the paralytic symptoms, and the question is whether these symptoms can be due to analogous growths in some other part of the nervous system. It is well known that tumors of the nerve trunks are common in association with neuro-fibromata of the skin. We have examined all the peripheral nerves that are palpable, but have been unable to determine any nodules upon them or any general enlargement. Although this does not exclude such changes, it makes their presence doubtful. In this connection is it remarkable to what an extent the nerve trunks may be involved by multiple neuro-fibromata without any loss of their function. This is in contrast to the single neuroma which usually produces symptoms.

Neuro-fibromata may develop in connection with the nerve roots within the spinal column; indeed, the cauda equina is a favorite site for their occurrence. When they affect nerve roots above and grow to a sufficient size, they at times make pressure on the spinal cord. There are a number of such cases reported in literature, but the clinical picture which they present is that of a pressure myelitis due to the growth of a tumor. The reflexes are exaggerated and objective sensory symptoms are more or less prominent. Adrian,[1] who is an assistant in the surgical clinic at Strassburg, where von Recklinghausen is pathologist, has published an extensive review

[1] Beiträge zur klinisches Chirurgie, vol. xxxi, 1901, p. 1.

of the literature of neuro-fibromatosis, especially in relation to the complications which occur in this disease. Reference to this article and to his review of the subject, which has just appeared,[2] will show what various nervous symptoms can result from this disease. This is no more than would be expected when we remember that this overgrowth of connective tissue may occur on any of the cerebral spinal nerves as well as in all parts of the sympathetic system. There are cases which follow the typical course of locomotor ataxia and others in which the diagnosis of syringomyelia was made and still more common, those which show a variety of complex motor and sensory paralyses. I have, however, been unable to find a case quite analogous to the one which I have presented to you. This patient shows a gradual loss of power associated with fibrillary contractions, muscular atrophy, loss of the deep reflexes and marked electrical changes. The trouble first affected the legs and is now involving the arms. There are practically no objective sensory disturbances, although the patient has suffered from sharp shooting pains. The bladder is somewhat affected. If we exclude the lightning pains the symptoms suggest a form of progressive muscular atrophy.

In Zinno's case,[3] which he calls multiple fibromatosis of the spinal ganglia with amyotrophic lateral sclerosis and considers a new morbid entity, the condition is very complex. At autopsy there were found tumors of most of the spinal ganglia, which were so large in the cervical region that they pressed on the spinal cord; together with this there were also the ordinary findings in cases of amyotrophic lateral sclerosis. The connection between the two is not made clear, at least in the abstract. Clinically, the case showed spastic paralysis, with muscular atrophy and no sensory changes. The absence of disturbances of sensation with such widespread involvement of the intervertebral ganglia is very remarkable, and may throw some light on our case.

Haushalter[4] has reported a most interesting case of a boy of fourteen, in whom the nervous symptoms were so pronounced and the skin lesions so comparatively slight, that for two years the diagnosis vibrated between myelitis, multiple sclerosis, and finally an unusual form of syringomyelia, until the presence of a skin tumor was noticed. In this case there was a spastic paraplegia with marked sensory symptoms associated with deformity of the spinal column and chest. There was slight ataxia in the arms, internal squint of the left eye and slight optic neuritis on the right side.

CLASSIFICATION OF NEUROMATA.

"NEUROMA."

- TRUE.
  - Neuroma verum ganglio-cellulare
    - myelinicum
    - amyelinicum

  } The occurrence of true neuroma in which ganglion cells are absent is doubtful.

- FALSE.
  1. Circumscribed or Solitary Tumors, growing from the connective tissue of nerve trunks, or of the ganglionic enlargements of nerves.
     - Innocent } Fibroma, myxoma, etc. Cysts from liquefaction of solid tumors (myxoma). The clinical "painful subcutaneous tubercle" is included here.
     - Malignant } Sarcoma, spindle-celled, fibro-myxo-sarcoma; cysts from liquefaction of sarcoma.
  2. Diffuse overgrowths of the connective tissue sheaths of nerves and of ganglionic enlargements of nerves. Neuro-fibromatosis.
     - Diffuse and generalized fibromatosis of trunks of nerves ("multipleneuromata").
     - Plexiform Neuro-fibromata.
     - Cutaneous neuro-fibromata (molluscum fibrosum).
     - Elephantiasis neuromatosa (pachydermatocele).
     - Pigmentation of skin of nerve origin.
     - "Secondary malignant neuroma," being the sarcomatous transformation of one or other of the above.

     } Various combinations of these.
  3. Traumatic or division neuromata.
  4. Enlargement of nerves in leprosy, syphilis, tuberculosis.

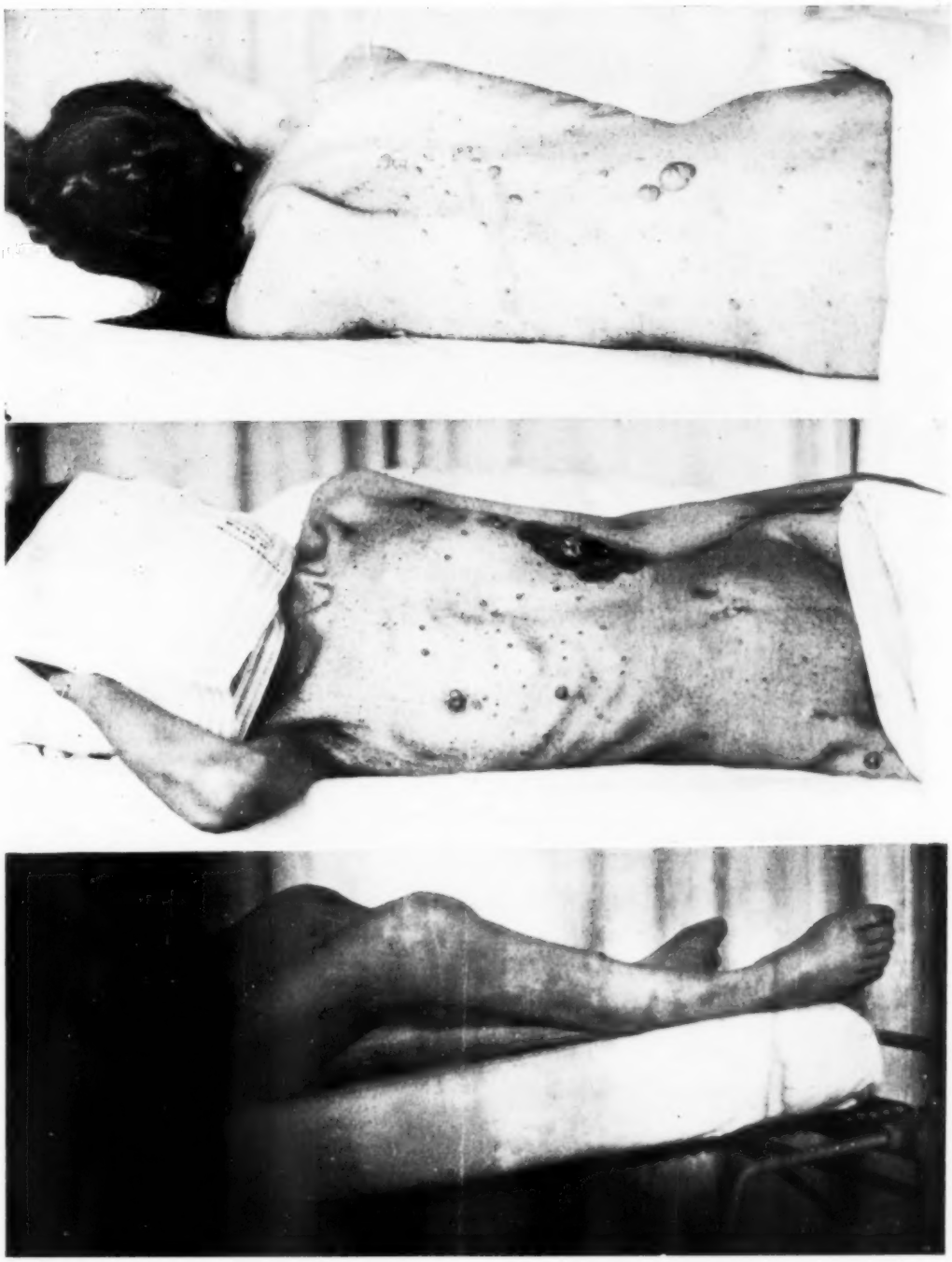
Sorgo's case[5] is still more instructive, as in his patient there were no skin lesions but very marked symptoms from the nervous system. A man of 47 had suffered for five years from intense pain on the right side of back and lower part of abdomen. There was for a time œdema over the lumbar spines which were painful on pressure and motion. Legs gradually became weak and flaccid with muscular wasting. The reflexes were lost. There were marked sensory changes which varied slightly from time to time. The bladder and rectum were paralyzed. The condition remained stationary for a year or more and then the legs gradually became spastic, the reflexes returned and became exaggerated. The sensory changes became more marked. Death followed the development of bed sores and cystitis. The diagnosis was tumor of the spinal

[2] Centralblatt f. die Grenzgebiete der Medizin u. Chirurgie, Bd. vi, 1903, Nr. 4, etc.

[3] Abstr. in Rivista Patologia nervosa e mentale, vol. ii, 1897, p. 524.

[4] Nouvelle Iconographie de la Salpêtrière, Tome xiii, 1900, p. 639.

[5] Archiv f. pathologische Anatomie und Physiologie, etc., Bd. 170, 1902, p. 399.



TO ILLUSTRATE DR. THOMAS' CASE OF NEURO-FIBROMATOSIS.

cord, probably cysticercus. At autopsy numerous tumors were found on the nerve roots of the cauda equina and other spinal nerves, together with two sub-dural tumors, pressing on the posterior aspect of the spinal cord. The larger tumor was between the XI and XII thoracic segments, and the smaller was at the level of the IX. They involved the nerve roots from these segments. There were no tumors on the peripheral nerves nor on the roots of the cerebral nerves. The tumors showed the structure of soft neuro-fibromata, and in the smaller ones it could be seen that the increase of connective tissue began around the individual nerve fibres and was arranged in concentric circles about them, forming an enormous overgrowth of their connective tissue sheaths. In the centers of these concentric circles of connective tissue were the axis cylinders of the nerve fibres, sometimes surrounded by their myeline sheath. The larger tumors were made up by the coalescence of the smaller tumors. In the spinal cord there was pressure myelitis with ascending and descending degeneration, and in the mid-sacral region there was, in the posterior horn, a tumor, which was called a perithelioma. Together with these findings, there was also a wide-spread change in the vessel walls, consisting of a great thickening of the intima and media. Sorgo believes that the flaccid paralysis was due to the neuro-fibromata involving the nerve roots, and that the spastic condition which subsequently developed depended upon the pressure myelitis, causing a condition of hyper-excitability of the reflex arcs. The fact that the axis cylinders persisted in most of the neuro-fibromata on the spinal roots explains why the deep reflexes could again make their appearance, after having been abolished. You may recall that quite analogous occurrences take place in certain tabetic patients, who suffer from a stroke of apoplexy and in whom the knee-jerk reappears in the paralyzed leg. Variability in the symptoms in neuro-fibromata of the nerves has been often noted. Thomson's sixth case, in whom there was a remarkable improvement in the paralysis, is a good illustration of this.

The condition which Sorgo describes of the great overgrowths of connective tissue around the individual nerve fibres recalls the description which Dejerine and Sottas [6] gave of the enlarged nerves in the case which they describe, under the name of "progressive hypertrophic interstitial neuritis of children," and one cannot help wondering whether these cases should not also be brought into connection with the already overburdened disease "neuro-fibromatosis."

The symptoms which these patients showed are a combination of those characteristic of tabes and of progressive muscular atrophy. The peripheral nerves are enlarged and easily to be felt.[7]

Although the symptoms in these cases differ in many respects from those of the patients whom you have seen, they show us what a wide range of nervous symptoms may occur in this remarkable disease, and the post-mortem findings explain why this should be. Indeed, it is not easy to think of any combination of symptoms which might not occur, for every part of the peripheral nervous system may be involved and in every imaginable combination. And nearly all parts of the brain and spinal cord may also be implicated.

In our patient we have a condition which, for the most part, appears to be due to a slowly progressing but a wide-spread involvement of the lower motor segment, and to explain this we can think of neuro-fibromata growing on the anterior roots, those which make up the cauda equina having been first and most affected, while the cervical roots have only lately begun to be involved. It seems strange that with such pronounced motor symptoms we have such comparatively slight sensory phenomena, and although there have been cases described in which the tumor formations appeared to affect only nerve roots of a similar function, it has been the sensory roots that have been selected. Our patient has suffered from the first from shooting pains and in spite of the fact that we have been unable to demonstrate any objective sensory disturbances, I believe that the posterior roots are also involved. You will remember that in Zinno's case there were no objective sensory disturbances, although all the intervertebral ganglia were involved in tumor formations. And in general, objective sensory disturbances are not very common unless the cord itself is implicated.

[6] Comptes rendus hebdomadaires de la Societe de Biologie, Tome v, 1893, p. 63.

[7] Dejerine: Revue de Medicine, Tome xvi, 1894, p. 881.

STUDIES IN TYPHOID FEVER.

SERIES I-II-III.

The papers on Typhoid Fever, edited by Professor William Osler, M. D., and printed in Volumes IV, V and VIII of The Johns Hopkins Hospital Reports have been brought together, and bound in cloth.

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THE CURE OF THE MORE DIFFICULT AS WELL AS THE SIMPLER INGUINAL RUPTURES.

By W. S. Halsted, M. D.,

Surgeon-in-Chief, The Johns Hopkins Hospital.

This communication will, I hope, be of interest to friends who have asked for precise information as to the modifications which our operation for hernia has undergone in the process of development during the past thirteen years, and of service to operators who seek to obtain in each instance a result as perfect as possible and who recognize that not infrequently there occur cases of hernia requiring for their cure extraordinary operative procedures. The present operation has been evolved by degrees and stands for the experience of 14 years derived from more than 1000 operations for the cure of inguinal hernia; features of the old where they seemed unnecessary have been dropped and new ones, as they seemed to be indicated, added. To record even the cruder general results of so many operations (upon adults with few exceptions)[1] for the cure of inguinal hernia are required special training, some zeal and a particular honesty of purpose; and for the recognition and interpretation of the nicer facts, keen perception and fine tactile sense are indispensable. A few drops or even a dram of fluid in the tunica vaginalis might readily escape detection, and to determine slight swelling or induration here and there in the epididymis and the relative size of the two testicles may be difficult. A novice can usually discover a distinct recurrence and so can the patient, but I have known an eminent surgeon to overlook a weakness in a scar of his own making sufficient to constitute, without doubt, a recurrence. The surgeon is fortunate and likely to be true to himself whose observations are controlled by mature assistants with large experience in the operative treatment of hernia and who are as eager as he to ascertain and state the exact truth.

If our operation for the radical cure of inguinal hernia has improved, it is due in no small measure to the arduous labors of Dr. Bloodgood, whose valuable contribution[2] should be better known. He established several facts of prime importance from his study of our first 300 cases of inguinal hernia. The majority of inguinal ruptures are now easily and quite well cured by a variety of procedures and by the average operator, hence it is difficult for the student and young practitioner to comprehend that it is hardly more than a decade since this variety of hernia completely baffled the efforts of the best surgeons to cure it. That so simple an operation as Kocher's can cure perhaps many of the milder ruptures, provided the neck of the sac is not too wide, leads to the inquiry whether the features, of these operations, upon which most stress has been laid may not be relatively unimportant, since operations of the magnitude of Bassini's and the author's are not in all cases indispensable. If the transplantation of the neck of the sac can cure so many cases, is it not possible that the transplantation of the cord, which at first was deemed so essential by Bassini and the author, may have owed its success in part to the fact that it made possible this very high closure of the sac's neck? Although for several years our operation, so far as transplantation of the cord and high closure of the sac is concerned, was even more radical than Bassini's (the cord was transplanted into the substance of the divided internal oblique muscle), we were tempted, at the very outset, to test the relative value of cord transplantation in some of the cases, and permitted the entire cord to lie undislocated and altogether undisturbed in its bed and to trust to the suture of the internal oblique muscle to Poupart's ligament, to the "lining of the wound with muscle" to effect a cure. It was well worthy of note, as Bloodgood emphasizes in his article, that all of the cases treated in this manner (cord undisturbed) remained cured. Another fact which Bloodgood's painstaking study established was that of one hundred and nine cases in which the larger bundle of veins of the cord was excised and the healing was per primam, not one showed a recurrence or any weakness at the site of the transplanted vas deferens, whereas in 6.4 per cent of the cases which healed by first intention and in which the veins had not been excised, there was a recurrence at the upper angle of the wound, at the site of the transplanted cord. And even in the wounds which suppurated, there was not a recurrence in the nine cases of vein excision, whereas, of eleven suppurating cases in which the cord-veins were not excised, four (36.3 per cent) recurred. In 118 cases, therefore, in which the larger bundle of veins was excised there was no recurrence at the site of the transplanted cord whether suppuration[3] occurred or not. And, certainly,

[1] The value of an operation for the cure of inguinal hernia can hardly be determined upon children for the surgeon is greatly assisted by nature as the child develops, and he is not confronted with the more difficult problems arising from an undeveloped or an acquired atrophy of the conjoined tendon, or from fatty degeneration and atrophy of the internal oblique muscle. Furthermore, the recurrences have almost invariably followed operations for the cure of very large and old ruptures, such as are impossible in children. And to quote from Bloodgood, "As we have had no recurrences" in children "whether the veins have been excised or not, it does not seem to make much difference what is done with the very small cord."

[2] Johns Hopkins Hospital Reports, vol. vii.

[3] Nine suppurations in 118 cases, and for most of which the author was personally responsible, seems a large percentage (7.6%) even for hernia cases ten years ago, but it was considered a good showing in those days. Since every one, including the operator, has invariably worn rubber gloves, suppurations even in the operations for hernia, has occurred in probably less than 1% of the cases. In 1890, all the assistants at an operation, the nurses and physicians, systematically wore gloves, but the operator wore them only for special operations, such as exploratory laparotomies, explorations for foreign bodies, loose cartilages, etc., in the joints, suture of the fractured patella, etc.—in other words, when there was a possibility of doing serious harm and no certainty of doing great good. By degrees the operator wore gloves more frequently, until Dr. Bloodgood as Resident Surgeon, and who had become thoroughly accustomed to them as assistant, wore them invariably as operator and demonstrated from our statistics the necessity of doing so. It seems to be a fact that one who has been trained to operate always

the cases in which the veins were excised, were not the simpler ones.

One of the most important of the facts ascertained by Bloodgood was the great variation in the width of the conjoined tendon and the responsibility of the insufficient tendon for the recurrences at the lower angle of the wound, through the external ring, direct. The transplantation of the rectus muscle recommended by Bloodgood [4] to close this defect seems to accomplish what its originator hoped it might, although, a priori, one would fear that this powerful straight muscle must eventually draw away from Poupart's ligament to which it had been sewed. Is it not conceivable, however, that a new encompassing fascia may develop about a transplanted muscle and that this fascia may remain even after the muscle has been pulled away? Experiments upon animals to determine this point would be interesting. M. Holl, now Professor of Anatomy in Gratz, directed attention many years ago to the part muscles probably play in the determination and development of the fasciæ.

Hence, so long ago as 1896 we recognized, thanks to Bloodgood, the value of the excision of the veins of the cord and the necessity for paying more attention to the neglected lower angle of the wound. Naturally, it was primarily to the upper angle that we had devoted our thoughts, for, as emphasized in one of the author's articles on the subject, "the cord is the first cause of the hernia and the ultimate obstacle to its cure." And this is true, notwithstanding the fact that recurrences at the lower angle were at first not very rare; for, our attention having been called to these lower angle recurrences, methods to cure them were soon found.

The success attending excision of the veins (one hundred and eighteen cases without recurrence at the site of the transplanted vas deferens) seemed to justify a continuance of this practice, provided it occasioned no undesirable results: but excision of the veins with transplantation of the vas deferens taught us that, not infrequently, a hydrocele, usually insignificant in size, was to be expected, and that in about 10 per cent of the cases atrophy of the testicle had occurred. Atrophy of this organ, however, was observed only in cases complicated by a very considerable swelling of the epididymis, and this observation of Bloodgood's, made so many years ago, has been verified by our study of more than one thousand operations. Great care was exercised, therefore, in excising the veins and, for a short time, a few months perhaps, this procedure was not so invariably practiced by all of us, being reserved for cases which seemed imperatively to demand it. We formerly handled the cord as, I presume, almost everyone still does; separated it, more or less roughly, by tearing, from the sac and its enveloping membranes, and raised it on a hook or strip of gauze preparatory to transplantation and while the stitches were being applied. We now treat the vas deferens with great deference, thanks again to Bloodgood. (Vide description of operation below.)

It occurred to Bloodgood before the publication of his report on hernia that it might be well to split the cord, transplanting only the veins to the outer angle of the wound and permitting the vas deferens to lie undisturbed. This method was finally abandoned by Bloodgood and other members of the staff who had practiced it, because the subtraction of the vas deferens did not appreciably reduce the size of the cord; furthermore, there were one or two recurrences at the site of the transplanted veins. This is a particularly good confirmation of the author's belief that the veins are largely responsible for the development of oblique inguinal hernia. The vas deferens contributes, relatively, very little to the size of most adult cords, but the veins, which at one moment make a bundle as large as one's finger, may the next and when empty be reduced to the size of a small quill. Is not this variation in the size of the cord possibly a factor in the production of hernia? When the hernia is first developing and the sac is, at operation, inside the internal abdominal ring, it can readily be demonstrated by a little pull on the veins. The fat, too, which is recognized as sometimes a probable factor in the production of hernia, accompanies for a short distance the veins rather than the vas deferens. This fat when present should be excised with the veins. For several years, then, we have been excising the veins in this careful manner, leaving the vas deferens untransplanted, undisturbed, and the internal oblique muscle undivided. In a few cases, however, without, that I am aware of, ultimate damage to the testicle, we transplanted the vas deferens to the outer angle of the wound. But we are quite certain that, as a rule, the less the vas deferens is manipulated and the more carefully the veins are excised, the less is the subsequent congestion of the epididymis. It is instructive from day to day to study the stump of the veins, the epididymis, the testicles, etc., after operations for hernia.

It is not the purpose of this communication to give the results in detail of these observations.

In a recent private case, urethritis Neisseri made its appearance a few hours after the operation. We naturally watched the epididymis on the operated side with some concern, fearing that excision of the veins might lower the resistance of this organ. On the twelfth day, without warning, a very slight induration of the epididymis became evident. I attributed

in rubber gloves finds it awkward to operate without them. I have more than once heard my assistants, while performing some insignificant operation without them, call for gloves because, as they said, they were conscious of unnatural finger movements, of a certain clumsiness without them. With gloves one probably acquires special methods of tying knots, holding instruments, etc. In our clinic the heavier gloves are exclusively used, although probably every member of the Staff has by predisposition been in favor of the thinner gloves and had to convince himself by trial of the thinner varieties that the thick ones, even with seams on the fingers, were preferable. The thin gloves were too slippery; also too unsafe, chiefly because of the danger of minute undetected holes. Cotton gloves, if changed very frequently, are undoubtedly better than no gloves at all. If the operator desires the physical property of the cotton which enables him to hold more securely and handle with more precision the intestines and viscera he might wear a very delicate gauze-mesh glove over the rubber or over two or three fingers of the rubber glove. Possibly a rubber glove might be manufactured with a wide gauze mesh permanently imbedded in its palmar surface.

[4] Anton Wölfler, Beiträge zur klinischen Chirurgie (Festschrift f. Billroth), 1892.

this to the fact that the patient carried out his irrigation-treatment badly, for the proper[5] method of irrigation being instituted, the swelling of the epididymis immediately subsided and the urethral discharge promptly ceased.

Four years ago the author used, for the first time, a part of the aponeurosis covering the right rectus muscle to close the lower part of the right inguinal canal. I felt compelled in this case to resort to some such measure, for the internal oblique was fatty and attenuated to a degree not very often seen by us, and the rectus muscle did not seem to promise so much as its fascia did. This patient was a college-mate of mine and for this reason I wished, perhaps, more than ever, to be very sure of the result. One year ago I examined this patient very carefully and was gratified to find as solid a closure as one could desire. I considered the result as perfect as any that I had seen. Dr. Harvey Cushing, house surgeon at the time, made a sketch of this act of the operation, which Brödel has kindly elaborated (vide Fig. VII). This procedure may have a wider application than I have proposed for it. The anterior sheath of the rectus muscle might be employed in the way described whenever the conjoined tendon is insufficient, whether the cremaster muscle can be well used to remedy the defect or not. And Berger[6] has recently suggested using the rectus sheath in much the same way in operations for the cure of inguino-interstitial hernia.

In the upper part of the canal we have strong tissues and plenty with which to close, and hence it was perhaps natural to transplant the cord to the upper angle, to bring it out through thick muscle. But it is not perfectly certain that the cord may not be a useful adjunct in the closing or filling in of the lower angle in some cases, and it is a fact that with Bassini's operation the percentage of recurrence at the position of the transplanted cord in the case of adults has been quite large, probably over 6 per cent. Whatever the truth may be, we have in the excision of the veins a distinct contraindication to transplanting the vas deferens, and thus far we have had no reason to believe that the results would have been better if the vas deferens had been transplanted, as was our custom for several years, to the outer angle of the canal. We may eventually discover that the transplantation of the cord, which Bassini, and at one time the author, considered not only so important, but perhaps the principal feature of the operation, is harmful rather than helpful. Briefly, we may find that not only the vas deferens, but even the entire cord, would be more safely transmitted at the lower angle of the deep wound than at the upper. It would require a very large number of observations to determine this point because the percentage of recurrences is so small in these days; and it is unfair to compare the results of various operations in the hands of various operators. Surgeons do not seem to be agreed even as to what shall constitute a recurrence, or wound suppuration, and, if they were agreed, the personal element would still count for much.

The Use of the Cremaster Muscle.—A device which we hit upon in our efforts to close more securely the lower part of the canal, but which we now make use of as often as feasible, probably in over 75 per cent of the cases, is the utilization of at least a part of the cremaster muscle, which we formerly cut away. This is a step of the operation to which one is irresistibly drawn in some cases by the great strength of the cremaster and the firmness and extent of its attachments to Poupart's ligament. A natural insertion, such as this, of the cremaster and its fascia into Poupart's ligament, has in each case a value which can be demonstrated on the operating table and can be counted upon definitely to contribute something, and occasionally perhaps a great deal, to the strength of the abdominal wall; whereas the artificial insertion of the internal oblique into Poupart's ligament, although undoubtedly of the utmost importance and always to be tried for, may occasionally and perhaps often fail, from insufficient muscle, too great tension, or gradual redressment, to close securely even the upper part of the canal. The lower part of the canal, ordinarily protected by the conjoined tendon, can rarely be entirely safeguarded by the muscle fibres of the internal oblique when its conjoined tendon is deficient. The cremaster, on the other hand, seems in just these cases to serve a particularly good purpose. The cremaster, unaided, has repeatedly made such a complete and strong looking closure that we have felt the hernia would be well cured if the operation were abandoned at this stage.

I have today, June 10, 1903, examined a patient whose very wide inguinal canals (the gap would have admitted the hand) were closed eighteen months ago solely by the cremasters stitched over instead of under the internal oblique muscle; the result, in the opinion of those who examined the case, is absolutely perfect, on both sides. My house surgeon, Dr. Fol-

[5] When the author's method of treating gonorrhœa can fail in his own wards, because improperly understood, it is not strange that so admirable a surgeon as Dr. Orville Horwitz, apropos of Janet's work on the abortive treatment of gonorrhœa by permanganate of potash, should write: "In spite of the claim of quick cures and prevention of complications a length of time elapsed before it began to be generally adopted in this country. The profession was skeptical as to the claims made for its brilliant results. This was probably due to the disappointment which had followed the employment of retroinjections of hot water suggested by H. Holbrook Curtis, and of the continuous irrigation with a hot solution of mercury bichloride, recommended by Dr. W. S. Halsted, which at the outset seemed to offer more benefit to the patient than the conservative methods then in vogue, but resulting after a fair trial by a large number of observers in being found valueless and often dangerous; the employment of these remedies having been found to be attended with great discomfort to the patient and being frequently accompanied by severe complications, such as acute posterior urethritis, seminal vesiculitis, prostatitis, and cystitis." This is not the proper time to tell how one must use the bichloride solutions in order to obtain the best results which have been claimed for it, but to judge from my own experience with this method twenty years ago in private practice, too much has hardly been said in its favor. The bad and indifferent results probably come from mismanagement or misconception. I should be glad at some future time to publish the treatment in detail, for it happens that I have not heretofore described or, in print, claimed anything for the method which rightly bears my name. I agree with Dr. Horwitz that irrigation with hot water is not only useless but dangerous.

[6] P. Berger: La Hernie inguino-intersitielle et son traitement par la Cure radicale. Revue de Chirurgie, Janvier, 1902.

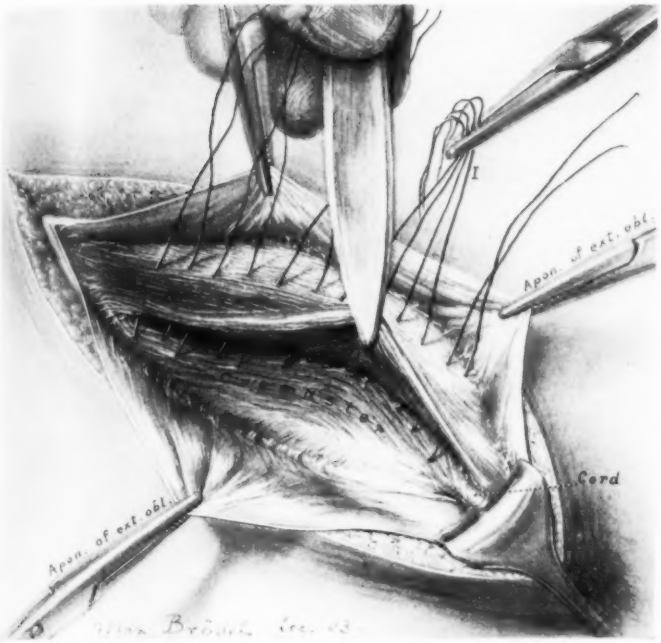

FIG. I.

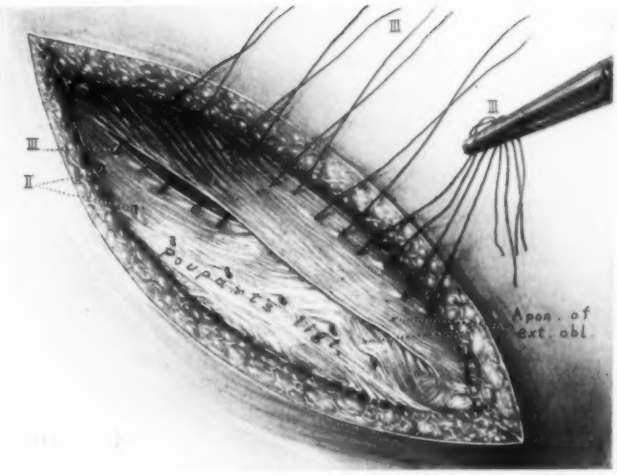

FIG. III.

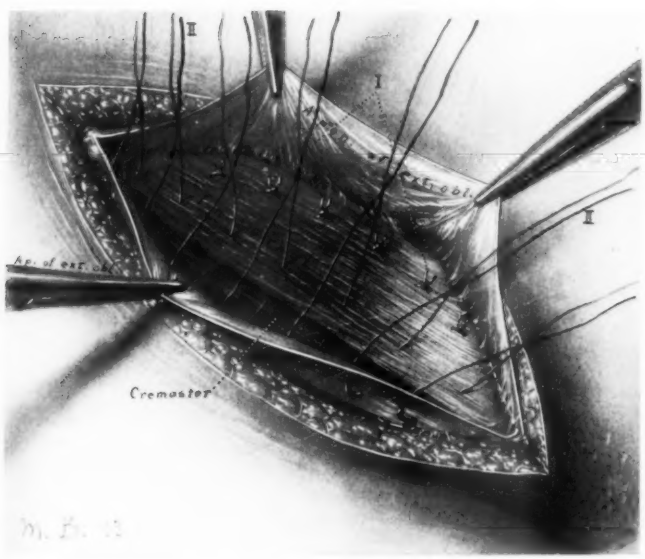

FIG. II.

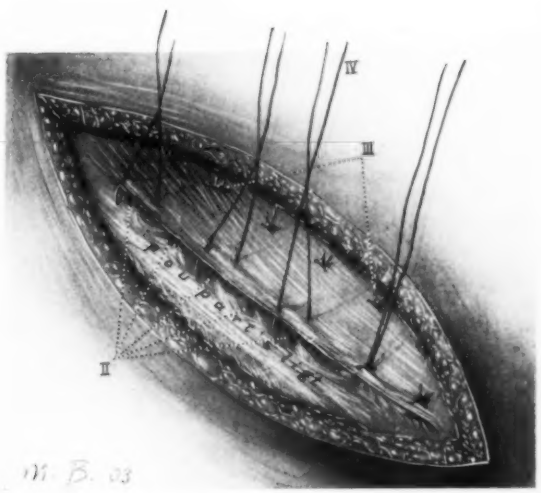

FIG. IV.

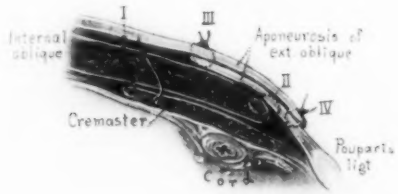

FIG. V.

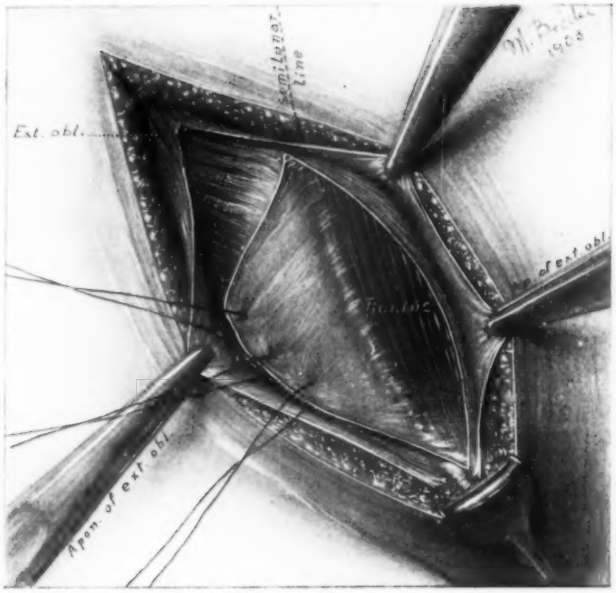

FIG. VII.

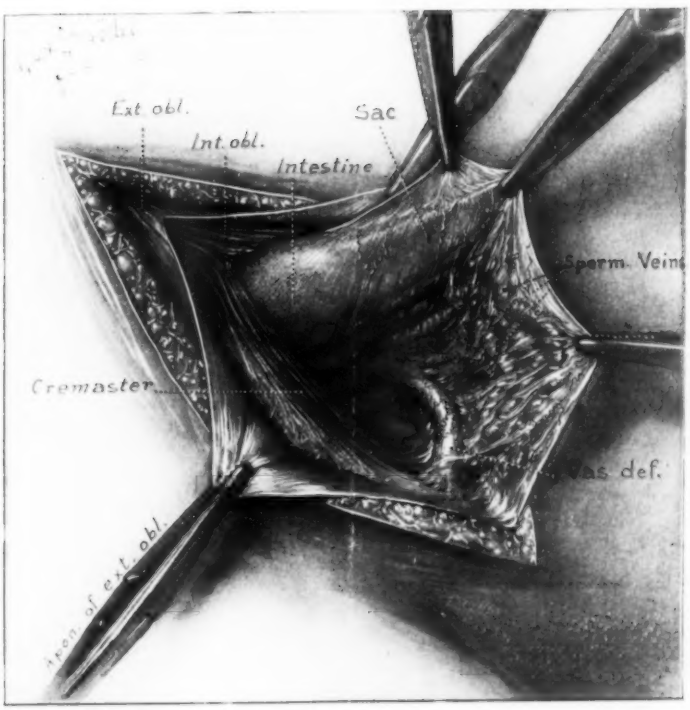

FIG. VI.

lis, and one or two others examined the man[7] with me. Even had I known what the result in this case was to be, I would have used, if possible, the internal oblique muscles in the old way, and hence have stitched the cremaster under rather than over the former. But the muscles were attenuated and not close at hand. Stitching the cremaster over the internal oblique muscle necessarily precludes the sewing of the latter to Poupart's ligament. The closure with the cremaster seems almost ideal in some cases; it is a method so inviting during the operation, and so true, when finished, to one of the *great principles of surgery; there is no tension.* It is, in this respect, as a plastic operation should be. What the ultimate verdict will be it is too soon to predict. The cremaster fibres, particularly the hypertrophied ones, will, in time, atrophy; but when this occurs, the cremasteric fascia, perhaps stronger than before, would probably remain, holding together the atrophied muscle bundles. There can, at least, no harm result from this attempt to strengthen the wall, for the internal oblique muscle has been used in the usual manner. The worst that could happen would be a recurrence, in a certain class of cases, at the lower angle, one that might, possibly, have been avoided if the aponeurosis over the rectus muscle had been employed instead of the cremaster as described by the author. The future will decide these nicer points, and it would seem that only the nicer points remain now to interest the operator.

Another feature of the present operation is to transplant the neck of the sac as described below. It is merely an additional precaution warranted by the good results obtained by Kocher and others with his operation.

And, finally, we overlap the aponeurosis of the external oblique muscle to insure the union which a mere approximation of the edges of the aponeurosis cannot do, and to close more snugly the external ring.

We still examine with the same care, but no longer with concern, the epididymis, testicle, stump of veins, etc., chiefly to ascertain if there is congestion (induration) of the epididymis or fluid in the tunica vaginalis. Often there is an appreciable, though very slight, induration of the epididymis, particularly if the veins have been ligated through the dense plexus very near the testicle; and often a few drops or a drachm or two, or even more fluid is present in the tunica vaginalis. This may become absorbed in a few weeks or months and might, when present, usually not be noticed by the patient except for the repeated careful examinations. Hydroceles containing several ounces have been recorded in our histories; in two or three instances operation for the cure of the hydrocele has been performed. What the proportion of hydroceles is to the cases operated upon for the cure of hernia, without vein excision, I cannot say for the reason that we excise the veins almost invariably nowadays, and in the days when the veins were not excised we did not observe our cases quite so keenly with reference to this point. One of the larger hydroceles followed, as I have said, an operation in which the veins were neither excised nor transplanted nor in any way disturbed. The patient, a navy officer, had an indirect rupture on each side. Both sides were operated upon at the same time and on both, hydroceles developed in a few days, although neither epididymis became more than just perceptibly indurated; but the larger hydrocele was on the side of the undisturbed veins and of the smaller hernia. Not a single atrophy of the testicle has been recorded since 1899, when Bloodgood published his report, and I believe that at that time it was noted that not one had been observed for several years.

Possibly some of my readers will ask, "Why take so much trouble, why make the operation so complicated when such good results as are published may be obtained by simpler methods?" The operation is not complicated for the surgeon competent to operate for the cure of hernia, nor are all its details required for the simpler cases, and we do not know just what the results obtained by simpler methods are. We cannot ascertain definitely even our own results, although we make a great effort and are admirably equipped to do so. This can be said, however, that, since the publication of the author's second paper, June, 1892, not a single recurrence has been charged to him. One of the world's most distinguished surgeons, the inventor of a clever hernia operation, made, with reference to himself, some such remark to the author three or four years ago, and the next morning two recurrences presented themselves. This surgeon permits his patients to get out of bed in eight days because, as he said to me, "A man can better afford to be operated upon three or four times for recurrence by my method than once by a method like McEwen's, which requires lying in bed for five or six weeks." In my experience a man would, *after* operation, prefer to spend several additional weeks in bed than run the risk of a recurrence. It is only before, not after the operation that a patient objects so vigorously to the time to be spent in bed.

The Operation.—The several steps of the operation are so well depicted by the illustrations of Brödel that a verbal description is almost superfluous for those who have the plates.

(I) The aponeurosis of the external oblique muscle is divided and the two flaps reflected as in the Bassini-Halsted operation.

(II) The cremaster muscle and fascia is split, not directly over the centre of the cord, but a little above it.

(III) The internal oblique muscle is made as free as possible. A little artefaction is here often necessary. If the muscle cannot be drawn, without tension, well down to Poupart's ligament, it helps, I think, to make a relaxation cut or two in the anterior sheath of the rectus muscle under the aponeurosis of the external oblique muscle. This sheath being in part the

[7] The history is briefly this. Male, æt. 59 years; Surgical No. 12,905; was operated upon January 15, 1902, for two very large scrotal ruptures, eighteen and twenty-four inches long (from external ring to bottom of scrotum). The conjoined tendons on both sides were almost obliterated. The circumcentral rings easily admitted four fingers. The cremaster muscles, very well developed, were used to close the entire dehiscence because the internal oblique muscles could, only with great tension, be drawn down to Poupart's ligament; the former was stitched in front of instead of behind the latter muscles. We had never before and have never since had occasion to use the cremasters in this way. Dr. Mitchell, my house surgeon, operated upon one side, and the author upon the other.

aponeurosis of the internal oblique muscle, one can readily comprehend that incisions into it, if properly made, might be of service. It is well, however, to postpone making such incisions until the sewing of the internal oblique muscle to Poupart's ligament is begun, for then the amount of tension can be nicely gauged and the number, length and precise position of the relaxation cuts determined. A second reason for postponing the relaxation incisions into the anterior sheath of the rectus muscle is that we sometimes use this portion of the rectus sheath to close the lower port of the inguinal canal, as already stated.

(IV) When the veins are large, and this is usually the case, they should be excised with very great care to avoid even the slightest extravasation of blood into the tissues about the smaller veins and about the vas deferens which they accompany. And the vas deferens, as first emphasized by Bloodgood, should not be raised from its bed or handled or even touched, lest thrombosis of its veins occur.[8] (Vide Fig. VI.) The veins should be ligated as high up in the abdomen as possible, being pulled down quite firmly just before the ligature (in a needle with the blunt end first) is passed between them. As a precaution against slipping, we apply two ligatures of fine silk, both for the abdominal stump and for the testicle stump of the veins. The farther from the testicle the veins are divided, the better, provided, of course, that their stump is external to the external abdominal ring.

(V) Ligation of the sac by transfixion or by purse string suture at the highest possible point. Both ends of this suture, after tying, are threaded on long curved needles, then carried far out under the internal oblique muscle from behind forwards, and, passing through this muscle, about 5 mm. apart, are tied. The idea was suggested to the author by Kocher's operation, the principle being essentially the same.[9]

(VI) The lower flap of the cremaster muscle and its fascia is drawn up under the mobilized internal oblique muscle and held in this position by very fine silk stitches, which, having engaged firmly a few bundles of the cremaster, perforate the internal oblique, preferably where it is becoming aponeurotic, and are tied on the external surface of the latter; vide Fig. I.

(VII) The internal oblique muscle, mobilized, and possibly further released by incising the anterior sheath of the rectus muscle, is stitched (the conjoined tendon also) to Poupart's ligament in the Bassini-Halsted manner. (Vide Fig. II.) Catgut is usually employed for this suture. The drawing was made from an unusually muscular subject and possibly exaggerated the size and extent of the internal oblique muscle, as well as of the cremaster, although the artist endeavored to record accurately what he saw.

[8] The fact is that the vas deferens is frequently accidentally handled or squeezed, but harm that we know of has never resulted since we have recognized the necessity for exercising great care in the separation and ligation of the veins.

[9] I have read recently in the Centralblatt für Chirurgie a reference to some other surgeon's account of this very procedure, but, unfortunately, cannot recall the surgeon's name and have not the facilities at this moment to hunt for it.

(VIII) The aponeurosis of the external oblique muscle is overlapped, as shown in Figs. III and IV. This is known as Andrew's[10] method, although devised independently by us.

(IX) The skin is closed with a buried continuous silver suture, and the incision covered with five or six layers of silver foil. It is unnecessary to dress or examine a wound closed in this manner for two weeks, when the wire may be withdrawn. Patients are kept in bed from eighteen to twenty-one days.

We hope to be able to publish very soon the results of the first 1000 operations performed for the cure of inguinal hernia at the Johns Hopkins Hospital. Certainly more than two-thirds of the operations have been performed by my associates, Drs. Finney, Bloodgood, Cushing, Mitchell and Follis, for we are all much interested in the subject. Each operator has been at perfect liberty and is encouraged to perform the operation according to his best judgment. This fortunately furnished a little variety, but of late the operation has, in almost every detail, been performed just as the writer has described it.

Inasmuch as only a limited number of surgeons see the Johns Hopkins Hospital Reports, in which Dr. Bloodgood published his article, it may be well to publish one or two of the Summaries which he prepared with such care and so great labor. He intends quite soon to investigate the condition of all those, so far as possible, who are included in these Summaries.

"Summary of the Ultimate Results. Complete to June 1, 1899."

"Recent cases, less than 6 months, and cases lost track of were not included.

All cases Group I to V healing p. p.	301 cases.	13 recur.	4.3%
All cases Group I suppurating	31 "	9 "	29%
Total	332 "	22 "	6.6%
Halsted's operation, Group I, healing p. p.	218 cases.	9 recur.	4.1%
Halsted's operation, Group I, suppurating	20 "	6 "	30%
Total, Group I	238 "	15 "	6.2%

"Recurrence in Wounds Healing Per Primam."

	Cases.	Recurrences.
(1) At the position of the transplanted cord, veins excised	109	nil.
(2) At the position of the transplanted cord, veins not excised	109	7 (6.4%)
(3) Upper angle of the wound, cord excised or not transplanted	83	1 (1.2%)

[10] The Chicago Medical Recorder, August, 1895, vol. ix, p. 67.

	Cases.	Recurrences.
(4) Lower angle of the wound, conjoined tendon wide and firm, rectus muscle not transplanted.....	264	nil.
(5) Lower angle of the wound, conjoined tendon obliterated, rectus muscle not transplanted.....	8	5 (62%)
(6) Lower angle of the wound, conjoined tendon obliterated, rectus muscle transplanted	14	nil.
(7) Lower angle of the wound, conjoined tendon wide and firm, rectus muscle transplanted	16	nil.

"RECURRENCE IN WOUNDS HEALING BY SUPPURATION."

(1) At the position of the transplanted cord, veins excised	9	nil.
(2) At the position of the transplanted cord, veins not excised.....	11	4 (36.3%)
(3) Upper angle of the wound, cord excised or not transplanted.....	11	1 (9%)
(4) Lower angle of the wound, conjoined tendon wide and firm, rectus muscle not transplanted.....	27	2 (7.4%)
(5) Lower angle of the wound, conjoined tendon obliterated, rectus muscle not transplanted.....	4	2 (50%)

Dr. Bloodgood's "*Conclusions as to the operation for inguinal hernia,*" published in 1899:

"Our observations prove that Halsted's operation with the excision of the veins will give perfect results, except in those few cases in which the conjoined tendon is obliterated; in these cases our observations so far have demonstrated that the transplantation of the rectus muscle will give perfect results."

"If the veins could be excised in every case of inguinal hernia and the remainder of the cord transplanted without any risk of epididymitis and atrophy of the testicle, a perfect result would probably be accomplished in every case."

"The operation would then be: The ligation and excision of the veins, the transplantation of the remaining portion of the cord into the upper angle of the divided and transplanted internal oblique muscle, and, in cases in which the conjoined tendon is obliterated, the transplantation of the rectus muscle. So far we have not observed a single recurrence when these procedures have been adopted. The sole objection to this method is the danger of atrophy of the testicle after excision of the veins. Atrophy of the testicle has been observed only after a very marked epididymitis. The probabilities of this epididymitis are very much less when the veins are excised without disturbing the vas deferens and its immediate vessels. For this reason I should advise that when the veins are excised the remainder of the cord, a very small affair, be left undisturbed. I am very much inclined to believe that the cord, reduced to such a diminutive size by the excision of the veins, will be as little likely to be the cause of a recurrence in the lower angle of the wound as in the upper angle when it is transplanted."

"*Cases in which the Veins should not be Excised.*"

"When during the dissection of the sac the cord is torn from its bed in the inguinal canal and subjected to traumatism, and the testicle withdrawn from the scrotum, the veins should not be excised, because the probabilities of epididymitis and atrophy are too great. In such cases I would advise the transplantation of the veins alone, so that the larger cord is divided,[11] and the wound is weakened less by the presence of a very small cord in two places than by the presence of a larger cord in one place, which from our results we know to have been the cause of a recurrence in 6.4 per cent of the cases."

"Note, June, 1899. In October, 1898, I performed for the first time the splitting of the cord, transplanting the veins only. Since this date the modification has been followed in 26 operations for inguinal hernia. In 12 the rectus muscle was transplanted. The wounds in 25 cases healed per primam. In 19 cases no swelling of the testicle followed operation. In 7 cases there was slight but temporary swelling. Thrombosis of the veins was not observed in any of the 26 cases. It is seven months since the first two operations. Both are perfect results. The others are recent operations."

"When the bundle of veins is unusually large, and complete excision is contra-indicated for reasons already given, I have suggested that a portion should be ligated and excised and the remainder transplanted. This has been done in a recent case by Doctor Cushing."

"In children the veins should not be excised; the probability of atrophy is greater than in adults. As we have had no recurrences whether veins have been excised or not, it does not seem to make much difference what is done with the very small cord."

"In the female the round ligament and its vessels is such a small affair that it makes little difference what is done with it."

"References to the transplantation of the rectus muscle by Wöfler: Wöfler published his method of transplantation of the rectus in 1892 in the Beiträge z. Festschrift f. Th. Billroth. I did not see this publication until my colleague, Dr. Clark, returned from Germany, in June, 1898. My preliminary report had then just been published. For this reason no mention was made of Wöfler's work. In the Archiv für klinische Chirurgie, June, 1898, Dr. Slajmer publishes 150 operations after the Wöfler method. A careful reading of these two articles has convinced me that this method of transplantation of the rectus differs from mine. In the first place no special reasons are given for the transplanting of the rectus muscle, while in my publication the reason given for the transplantation of the rectus is to strengthen the lower por-

[11] The splitting of the cord has been discontinued by its author.

tion of the inguinal canal by the introduction of muscle which is weakened by the obliteration of the conjoined tendon. The description of the Wöfler method and the illustration on page 912 of the second article show that the rectus muscle is not transplanted in the best way to strengthen the lower portion of the wound, because the sheath of the rectus is not divided down to the symphysis pubis; but the division of the sheath ends at least 2 to 3 cm. above the pubic bone. For this reason the transplanted rectus muscle is approximated chiefly over the upper two-thirds of the wound. In addition, Wöfler divides the sheath of the rectus on the anterior surface above the linea semilunaris. In my method the sheath of the rectus is divided posteriorly and the belly of the muscle is brought out behind the internal oblique. I believe that by this method the muscle can better be transplanted so as to occupy the lower two-thirds of the wound. Slajmer reports 6 recurrences, about 6 per cent. In three of these cases the wound suppurated."

THE PATHOLOGY OF SMALLPOX.

By Wm. Royal Stokes, M. D.,

From the Laboratory of the State and City Board of Health and the Pathological Laboratory of the College of Physicians and Surgeons, Baltimore, Md.

A number of interesting changes have been noted in the various tissues and viscera of smallpox cases, and these changes are widely distributed throughout the system.

The pustular eruption is frequently found on the surface of the mucous membrane of the respiratory tract, and these often coalesce as ulcers, or masses of fibrinous inflammation.

These pustules and ulcers are often seen in the bronchi of the third and fourth order, and are quite frequent at the bifurcation of the bronchi. Necrosis of the surface epithelial cells is also very frequent, and fibrinous and purulent inflammation is also often present.

The lung often contains areas of broncho-pneumonia, and the liver, the kidney and the heart muscles show cloudy swelling and fatty degeneration. The heart muscle also often exhibits fragmentation of the fibres. The acute, soft splenic tumor of infection is usually present.

An interesting set of changes has been described by Weigert,[1] consisting in areas of local necroses in the liver, spleen, kidney and lymph glands. These areas consist of degenerated masses of coagulative necrosis containing much nuclear detritus, and many degenerated cells without nuclei.

The lesions of the bone-marrow first described by Chiari,[2] and called "osteomyelitis variolosa," are said to occur in 72 per cent of all cases. These small scattered nodules are about the size of a pea, and under the microscope they consist principally of epithelioid cells, which are probably produced from the normal marrow cells by proliferation. Leucocytes and traces of fibrin are also present, and the masses often undergo coagulative necrosis.

Chiari[3] examined the bone-marrow of 22 cases of smallpox in various stages with the following results: During the stage of simple early eruption, he investigated five cases, and found microscopic changes in three instances. Nine cases were observed in the pustular stage, of which eight gave positive results. Eight cases were examined in the stage of healing or scaling, and all gave positive results. In one of two cases who died from other causes about two months after recovery from smallpox, these necrotic masses were found.

The process is found especially in the yellow marrow of such bones as the femur, tibia, ribs, sternum, and the body of the vertebræ, and begins by a proliferation of the normal marrow cells. These form masses of cells, whose nuclei are surrounded by much cytoplasm. A few neutrophilic leucocytes are also found in these masses of epithelioid cells, and they soon show a central area of necrosis which often contains fibrin. He only demonstrated micrococci in one case as present in these necrotic masses.

Similar areas are frequently found in the testicle, and this condition is called "orchitis variolosa." In hemorrhagic smallpox the above changes are usually present in combination with local hemorrhages in the skin and various serous cavities.

Changes in the Skin.

The first change, according to Unna,[4] consists in an edema and swelling of the epithelium. Many of these cells undergo softening and colliquation, forming a cavity, while others remain as septa, dividing the cavity up into smaller loculi.

The epithelial cells undergo a special degeneration which is known as ballooning, and which affects all of the various layers, even penetrating into the hair follicles. These swollen cells often attain from two to three times their normal size. They sometimes retain their nuclei, but the cytoplasm becomes perfectly clear, and the nuclei are often fragmented. It is a variety of coagulative necrosis. Sometimes two or three nuclei are present in one cell.

The epithelial cells continue to undergo liquefaction, and this causes an increase in the size of the cavity, at the same time mitoses are found in the cells forming the circumference of the cavity. The cells become compressed, and undergo fibrinoid degeneration, forming strands in the cavity which give the reaction for fibrin.

Umbilication is explained by the fact that the degenera-

tion and edema of the epithelial cells take place more at the periphery of the vesicle. The less swollen centre remains behind. Unna does not believe that the cords of the compressed epithelial cells running through the vesicle act as guy ropes, but that the edema and compression is exerted more at the sides of the vesicle, and thus this portion of the lesion extends beyond the less compressed centre.

In the vesicular stage an exudation of serum takes place, and neutrophilic leucocytes also emigrate from the vessels into the vesicle forming the pus of the vesicle. These infiltrate the cutis, and also soon fill the vesicle, producing the pustular stage of the disease. This primary invasion of leucocytes is due to the smallpox poison, but often at the end of the first week there is a second leucocytic invasion, due to a secondary infection with the pyogenic cocci.

Healing.

As the pustule begins to dry, a scale or crust is formed, and the lesion is invaded by sprouting projections of newly-formed epithelial cells. The upper layers become horny, the lower layers form the other normal cellular layers. In order not to form a scar, the newly formed epithelial cells must grow in an even layer with the convex surface downward, and the stratum Malpighii must not have been destroyed by the process of degeneration.

Plasma Cells.

There is one change which should be mentioned in this connection, and that is the appearance of the plasma cells in the corium. It is held by many that the injury caused by the poison to the epithelial cells is the very first change which is apparent. Dr. Gilchrist, however, has kindly loaned me some sections made from very early papules, and although the epithelial cells show little or no change, the corium shows a marked infiltration with plasma cells. These are usually collected about small arteries and capillaries.

Dr. Gilchrist is of the opinion that this plasma-cell invasion is the earliest change to be noted in smallpox, and this opinion accords very well with the best conception of the method of infection.

It is inconceivable that the poison comes in contact with every portion of the skin at practically the same time, thus causing infection. It seems more rational to believe that the cause is inhaled, and that by means of the circulation it reaches the skin, where it causes widely distributed lesions.

The primary exudation of the plasma cells from vessels followed by the various epithelial lesions supports this theory.

Pathological Study.

The following pathological study was made from a series of five autopsies performed during the past year, and from sections and cultures in six non-fatal cases. Four of the cases were adults, and one case was that of a child 2 years old.

All of the cultures in the non-fatal cases were made from vesicles or early pustules, and in every case they remained sterile. Five sets of cultures were made from the autopsies, and in all of these cases the condition had advanced well into the pustular stage. Four cases gave a pure growth of Streptococcus pyogenes from the pustules, and one case of hemorrhagic smallpox showed the presence of Staphylococcus pyogenes aureus.

Various attempts were made to cultivate the specific cause of the disease. Material from bacteriologically sterile pustules was inoculated into eggs, on the surface of coagulated egg albumen, and in 1 per cent sterile milk, as this is said to be a good nutrient material for amebæ. All of the results were negative.

Pathological Changes in Smallpox.

In two of the five cases the respiratory tract was the seat of extensive changes. The inner surface of the larynx and trachea were both almost entirely covered with a dirty yellowish pseudomembrane. The under-surface of the epiglottis was also involved.

Histo-Pathology.

The primary condition consisted in an extensive degeneration of the lining epithelium. Often the limiting membrane of the nucleus was destroyed, and the chromatin was scattered as a fine dust through the cytoplasm, showing the condition known as nucleorhexis. Hyperchromatosis and irregularity in the size and shape of the nuclei also existed. These changes result in complete granular necrosis of the epithelium, and cause a necrotic layer without fibrinous deposits.

The submucous coat is often separated from the mucous coat by an exudation of fibrin and serum, and the former coat, together with the muscular coat, are richly infiltrated with plasma cells, small lymphocytes and cells resembling epithelioid cells. Even the cartilage cells show changes, consisting in the disappearance of the cytoplasm of the cell. The cell often forms an empty vacuole, with the nucleus forced to one side.

Streptococci in great numbers were stained in the masses of superficial necrotic cells, and in the vessels of the submucous coat.

Lungs.

The pleural surfaces of one case were dotted by vesicles about the size of a number six shot. These were very numerous, and on microscopic examination they seemed to be large lymph spaces distended with a serous fluid. One case showed a fibrinous pleurisy, and in three cases broncho-pneumonia existed.

In two of the cases the typical necrotic areas existed, which are so characteristic of smallpox. These consisted of a central area of coagulative necrosis containing numerous groups of streptococci. The necrotic centres were surrounded by a zone of cells, consisting of small lymphocytes, proliferated alveolar endothelial cells, and a few leucocytes. Both the necrotic area and the surrounding cells show masses of nuclear fragmentation, and many of the cells simply form a mass of granular debris. (See Fig. 19.)

The presence of actual bacteria in such large numbers in local necrotic areas is a departure from the usual rule, as in typhoid fever, eclampsia, diphtheria, and other diseases showing these changes, bacteria are not present necessarily, and the change is probably due to the toxin, or mechanical effects of cellular or fibrinous thrombi. Fig. 1 shows streptococci in an exudate in an air cell. Fig. 1 a shows the curious vesicles which were found on the pleural surface of both lungs in one of the cases of smallpox. At "a," two large distended clear spaces are seen, and "b" shows the normal lung tissue.

Heart Muscle.

In three cases the nuclei of the cardiac muscular fibres showed great irregularities in size, many being small and shriveled. Others were enlarged, irregular, oval or round, and stained more lightly than normal. In one case there was evidence of longitudinal splitting of the fibres, on cross section a central channel, containing radiating smaller connecting lines resembling a fine tooth comb, being present.

The Liver.

The livers in all cases were enlarged and yellowish, but very little fatty change was found microscopically. Cloudy swelling, the accompaniment of all acute infections, was always present. Under the microscope the enlargement was found to be due to congestion and cloudy swelling, and no fatty areas were seen. In one case a few collections of proliferated endothelial cells and small lymphocytes were present without actual general necrosis, and in another case necrotic areas, somewhat similar to those described in the lung, were detected. These two different varieties of necrosis of the liver correspond to those described by Councilman[4] and Mallory in their studies of the liver in diphtheria. The first variety they called disseminated necrosis, and these usually only consist of a collection of cells of various types. The liver cells present are necrotic and broken down, and the nuclei show various stages of degeneration.

Among the necrotic liver cells a few proliferated endothelial cells and leucocytes are usually found.

Although these changes were only made out in the liver of one of the five cases, the degeneration of the liver cells and the proliferation of the endothelial cells could be made out in these small areas, which in this particular case usually consisted of about twenty-five cells.

In one case, that of a child two years old, the so-called central necroses of Councilman and Mallory were detected in the liver. These usually occur near the central vein in diphtheria, but in one case of smallpox they were found in connection with the portal systems, and even in the middle of the lobules. They simply consisted of large collections of broken down necrotic masses of a homogeneous material resulting from the destruction of liver cells. These areas took on a brilliant red stain when treated with methylene blue and eosin. The necrotic areas are marked by large collections of nuclear detritus, and the destruction of liver cells can be well made out on the edge of the areas. The protoplasm of the cells coalesces into homogeneous masses, and in the early stages of destruction the chromatin of the nucleus breaks up into large irregular fragments, which are scattered through the cytoplasm of the cells. This was in a case of general streptococcus infection, but streptococci did not seem to play any important part in the production of these areas as particulate bodies. A few streptococci were found on the edge of these areas, but they were not detected in the middle of the necrotic material. Throughout the rest of the liver large groups of streptococci were found in the capillaries and spaces between the endothelial cells and liver cells. It would seem, therefore, that excluding the cause of smallpox, that these areas are caused by the soluble products of the secondary invader, the Streptococcus pyogenes.

Fig. 2 shows a typical area of central necrosis. At "a" the normal liver structure is seen, while "b" shows the outer zone of necrosis in which many liver cells can be made out in various stages of degeneration. Remains of the protoplasm and nucleus can still be made out. At "c" the central zone of complete structureless necrosis with intense nuclear fragmentation can be made out.

Microscopic Examination of the Kidney.

Extensive changes were found in every kidney examined.

In one case, in a child 2 years old, the acute interstitial nephritis, described by Councilman[5] in cases of scarlet fever and diphtheria, was found. The kidneys were enlarged, and the tubules were separated from each other by cellular infiltration. This infiltration also often surrounded the glomeruli outside of the capsule. These cells are not leucocytes, but are much larger, and the nucleus stains very deeply and is usually placed eccentrically at either end of the cell. Councilman believes that in some way these cells are derived from the small blood vessels, as they are often found within these vessels. He also thinks that their irregular shapes indicate that they are ameboid, and he identifies them as Unna's plasma cells. According to him they are large lymphocytes, and they emigrate from the vessels in acute interstitial inflammation of the kidney. They are very numerous in the spleen and bone marrow, and are probably found principally in these situations.

In one very malignant confluent case the kidney was the seat of most interesting lesions.

On looking at some of the glomeruli, with the low power, they seem partially changed into hyaline masses, and the high power shows that this condition is due to numerous large or small droplets of a clear hyaline material within the lumen of the capillaries. This condition is apparently due to an actual degeneration of the endothelial cells of the glomerular capillaries, since many of these can be seen in the various stages of degeneration. The nuclei seem to swell and take up a much paler stain than normal, and the cytoplasm breaks up into round hyaline droplets of various sizes, which often coalesce into large confluent drops, apparently obstructing and distending the capillary lumen.

Sometimes the chromatin of degenerating endothelial cells becomes increased in amount and granular.

The epithelial cells lining the capsular space also frequently proliferate, which compresses the capillary network into an irregular mass. They can be distinguished from the endothelial cells of the capillaries by their larger vesicular more lightly staining nuclei, and by the greater amount of eosin-staining cytoplasm. These proliferating capsular endothelial cells show a great tendency towards hyaline degeneration, which seems to begin by an increase in size of the nucleus, while the cytoplasm turns to round hyaline drops of various sizes. The nucleus takes on a paler stain with hæmatoxylin, and finally disappears in the hyaline material, which becomes confluent, forming large round or oval drops.

Sometimes the hyaline material forms a large crescentic mass of homogeneous clear material in the capsular space.

The epithelium of the convoluted tubules is swollen, and the cytoplasm of the cells contains numerous granules. Many of the cells, however, have undergone a much greater change. The cytoplasm is completely transformed into a mass of clear droplets of about the average size of from three to ten times the diameter of a micrococcus. The nuclei are usually well preserved, although the cells are often swollen to about twice their natural size, and simply consist of a mass of granules. These degenerated cells finally break up and the granules become free in the lumen of the tubule. Here they seem to coalesce into hyaline casts. The limbs of Henle are often distended and filled by casts of clear hyaline material. These areas of degeneration do not stain by Van Gieson's stain, and are therefore not true hyaline. When stained by Weigert's fibrin stain, however, both the clear droplets and the casts take up a deep purple stain. This is the reaction for fibrin, and the degeneration must be of a fibrinoid character. The formation of the hyaline casts from the degenerated cells can also be clearly made out, as many purple droplets are seen gradually coalescing to form casts taking the same stain. This is of interest concerning the somewhat doubtful origin of hyaline casts, and it would seem clear that in this instance they were formed from degenerated cells of the tubules undergoing fibrinous change. The formation of casts from somewhat similar large droplets has been described by Councilman and Mallory in diphtheria. These large droplets in the degenerated tubular epithelium of the cortex stain a deep blue color by Mallory's connective tissue stain, and the hyaline casts present stained also blue by this stain. Councilman and Mallory think that both the hyaline and granular casts are formed from these products of cell destruction.

When these degenerated cells are stained by osmic acid, and then counterstained by safronin, eosin or other stains, many of the smaller granules take up the black fat stain, but many remain colored by the safronin or eosin.

There are therefore fatty and fibrinous changes side by side in the same cell.

Fig. 3 shows streptococci in the liver.

Fig. 4 shows acute interstitial nephritis. In the centre of the photograph at "a," there is an atrophied, compressed glomerulus completely surrounded by the cellular exudation of plasma cells. This exudation also separates many of the convoluted tubules from each other.

Fig. 5 shows the degeneration of the epithelial cells of the tubules very well. The specimen was first stained by eosin, and then by Weigert's fibrin stain, and the cytoplasm is broken up into the large granules seen in the picture. These granules usually take up a deep blue stain by this method, thus giving the reaction for fibrin. The tubule marked "a" shows this degeneration, and at "b" a degenerated cell can be seen whose nucleus is still present. At "c" the tubule contains many desquamated, degenerated cells free in the lumen. Many of these cells have lost their nuclei, and some of them show the granules mentioned above. Fig. 6 shows a similar condition, and at "a" the tubule contains a granular cast which in the stained Weigert specimen takes up a light blue stain. It seems that these granules gradually coalesce into casts of a hyaline or granular nature.

CHANGES IN THE SPLEEN AND OTHER VISCERA.

The spleen was large and soft in two cases, and of normal size and consistency in three instances. Microscopically, nothing was found except acute congestion of the splenic spaces. The stomach, small intestines, large intestines, bladder, diaphragm, voluntary muscles, and pancreas were normal in those cases examined. The granular layer of the medulla of the adrenal gland in one case contained areas of cellular infiltration without necrosis. These consisted of small lymphocytes and cells of an endothelial type.

LESIONS IN THE SKIN.

No lesions of the skin were noted which have not been previously described, but the primary exudation of plasma cells has not been especially emphasized in Unna's description. These plasma cells are probably derived in part by proliferation from the endothelium of the lymph spaces and lymph vessels.

In some of the sections made from very early cases, the epithelial cells do not show any great injury, but the cutis is swollen, and there is an increased number of plasma cells in the lymph spaces, and around the small blood vessels. The condition resembles the response to some injury, and seems to be the first change in the skin, since the various changes in the epithelial cells are not yet present. Some of these cells have an eccentric nucleus, and resemble the cells found by Councilman in the hyperplastic bone marrow in diphtheria. Other cells in these groups exactly resemble the endothelium of small vessels, and are probably derived from lymph spaces and small vessels by proliferation. The typical plasma cells are thought by Councilman to be emigrated large lymphocytes.

Fig. 7 shows the primary cellular infiltration which seems to precede the injury to the epithelial cells. At "a" can be seen a normal layer of epidermis, and beneath this in the corium at "b" there is a large collection of the so-called plasma cells. Smaller groups are scattered through the cor-

ium at other points, and since the epithelial cells show no injury, this cellular infiltration seems to represent the primary lesion of the skin.

The next change in the earliest lesions was edema and swelling of the epithelial cells, followed by contraction of the nucleus. This often became fragmented and broken up into small granular masses. The exudation of leucocytes and serum, and the liquefaction of the epithelial cells was also observed.

The various changes in the epithelial cells are well shown in the photomicrographs. Fig. 8 begins a series showing the injury to the epithelial cells, and would correspond to the early papular stage of the eruption. At "a" various stages of nuclear atrophy and fragmentation can be made out, and at "b" the great edema and liquefaction of the cytoplasm of cells appears. This is the ballooning process of Unna. The process begins in the stratum lucidum and stratum granulosum. Fig. 9 simply shows a continuation of the same process. Fig. 10 shows a curious degeneration of the epithelial cells of the stratum granulosum. The nuclei are shriveled and the surrounding cytoplasm is broken up into circular clear masses resembling fat drops. At "a" the phantom-like outline of these globular, clear masses can be well made out, and this degeneration seems to be a further process in the formation of the vesicle. Fig. 11 shows a vesicle originally filled with a clear fluid. At "a," the junction of the stratum lucidum and the stratum granulosum, the layer of epidermis has split, and the layers pass above and below the fluid contents of the vesicle. Several hair follicles are seen at "b," and "c" shows that the corium is free from any participation in the vesicle. Fig. 12 shows a smallpox pustule, which has ruptured the lower enveloping layer of epidermal cells. Many neutrophilic cells have thus escaped, and have infiltrated the corium. Such accidents account for the scar formation following smallpox, or the so-called pitting, since the scar must form when fibrous tissue regenerates after inflammation. "A" shows the pustule which has ruptured at "b," and "c" indicates the corium richly infiltrated with neutrophilic leucocytes, and an edematous albuminous fluid. Fig. 13 is taken from a section made directly through the area of umbilication in a pustule. It shows very well that the umbilication is simply produced by an edema and purulent infiltration of the sides of pustule, causing a bulging, while the central portion of the pustule, which shows little edema, remains as a depressed centre. At "a" the layers of epidermis are separated by a purulent edematous fluid, and at "b" the exudation is entirely purulent. The central or depressed area of umbilication at "c" shows very little change, and at "d" the lower layer of epidermis has not ruptured, showing a normal subcutaneous layer at "e." The crater-like depression of umbilication is well shown. Fig. 14 shows a mass of interlacing filaments of fibrin, including pus cells within the meshwork. This exudation of fibrin is often seen in smallpox pustules, especially when there is a mixed streptococcus infection. At "a" the network of fibrin is seen ending abruptly at "b," the corium.

In early cases of smallpox a clear space forms around the nucleus, and this gives an appearance of a shrunken body inclosed in a clear capsule.

In the hemorrhagic case, the cells of the stratum granulosum showed various stages of nuclear degeneration, and the edema of the cytoplasm with typical ballooning was observed. The capillaries and lymph spaces of the corium were greatly distended by red blood corpuscles, and numerous hemorrhages were present in the connective tissue. The epidermis was not invaded by red blood corpuscles.

Changes in the Testicle.

Necrotic changes were noted in the testicle in two cases, and the various stages could be well traced out. The process seemed to begin by a necrosis of the epithelial cells of the seminiferous tubules. The nuclei of these cells showed marked karyorrhexis, and the protoplasm of the many cells was necrotic and granular. In the later stages, large areas of necrosis can be seen to particularly consist of seminiferous tubules which have coalesced into masses with central areas of coagulative necrosis, with nuclear fragmentation, surrounded by a thick zone of necrotic cells. The intertubular connective tissue is thickened, and even the walls of the arteries have undergone necrosis with nuclear fragmentation.

Fig. 15 is a good representation of the necrotic areas found in the testicle. At "a" a few normal seminiferous tubules are present, which at "b" are gradually undergoing necrosis, with loss of contour. The area marked "c" consists of a necrotic mass of coagulative necrosis with much nuclear fragmentation. Fig. 16 at "a" shows the seminiferous tubules under a higher power, and many of the epithelial cells show fragmentation or loss of their nuclei. The gradual transition of these injured tubules into necrotic material is also well shown.

Secondary Bacterial Infection.

This is a point of general importance, as the rapid advance of serum therapy towards a remedy for combating septicemia bears directly upon this matter.

It is now well known that many typhoid cases, and perhaps the majority of the eruptive fevers, die not so much from the primary cause, as from secondary infection with Streptococcus pyogenes.

This streptococcus infection is the most striking feature of fatal smallpox, and the course of infection can be traced from the skin to the general circulation.

As mentioned above, the cultures from vesicles in early and mild cases of smallpox remained sterile, but after the secondary fever the pustules often contain streptococci in abundance.

Four of our fatal cases after death contained streptococci in the pustules, and in the various viscera, such as the heart, liver, kidney, spleen, lymph glands, pancreas and lung.

On staining by Weigert's stain for bacteria, streptococci could be demonstrated in the pustules of the skin, and in the lymph spaces of the corium.

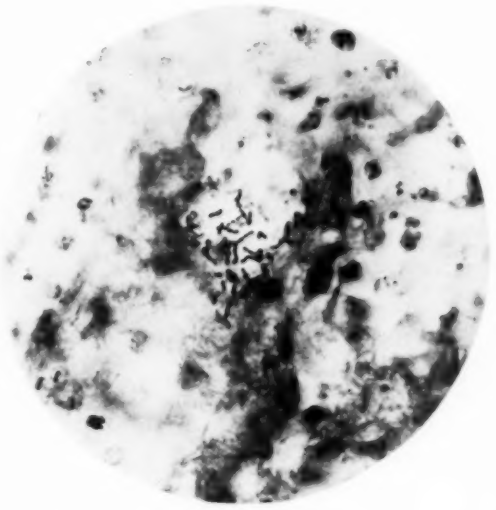

Fig. 1. Section of lung of smallpox case showing a purulent exudation in an air cell containing streptococci.—*Dr. C. H. Potter.*

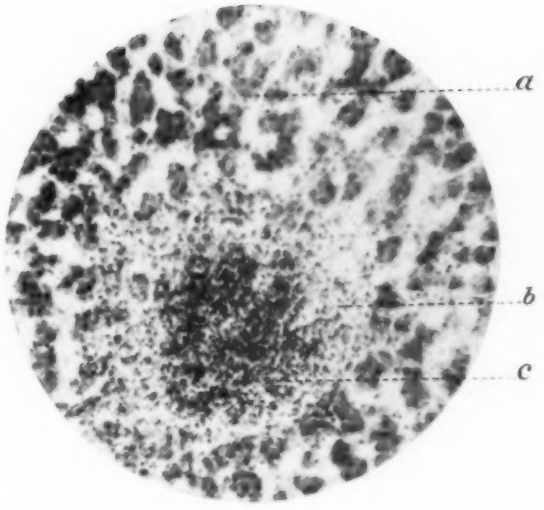

Fig. 2. Section of liver showing a typical area of central necrosis. The zone at "a" shows the rows of liver cells separated by capillaries, and "b" shows the outer area of necrotic liver cells in various stages of destruction. At "c" the complete structureless zone of necrosis can be made out.—*Dr. C. H. Potter.*

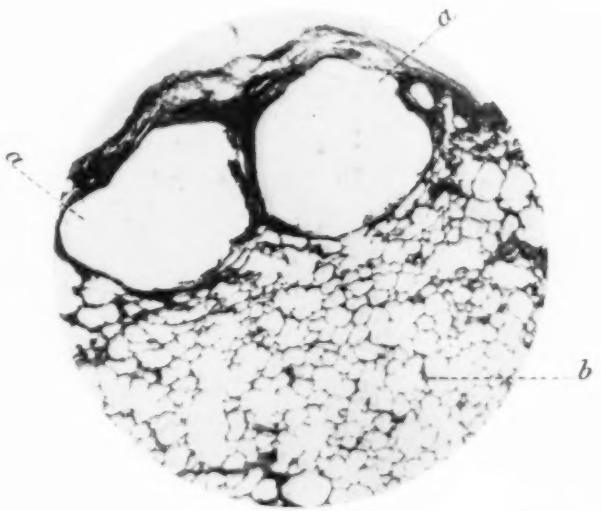

Fig. 1 a. Section of lung and pleural surface showing large spaces on the pleural surface filled originally with fluid. These pseudo-vesicles of the pleura are shown at "a," and "b" indicates the normal lung tissue.—*Dr. C. H. Potter.*

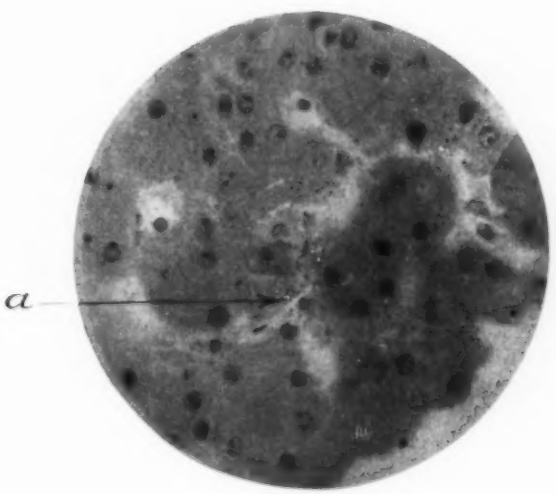

Fig. 3. Section of the liver showing a few streptococci at "a" in the capillary spaces of the lobule.—*Dr. J. S. Fulton.*

Fig. 4. Section of kidney showing acute interstitial nephritis under a higher power. At "a" a large group of plasma cells are seen surrounding a glomerulus and separating several tubules from each other.—*Dr. C. H. Potter.*

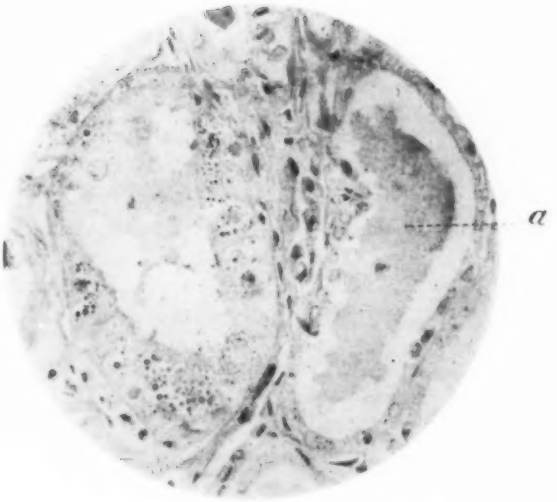

Fig. 6. Section of the cortex of the kidney showing the same degeneration as Fig. 5. At "a" a cast which stained by the fibrin stain is seen, showing the formation of the casts from these granular bodies.—*Dr. C. H. Potter.*

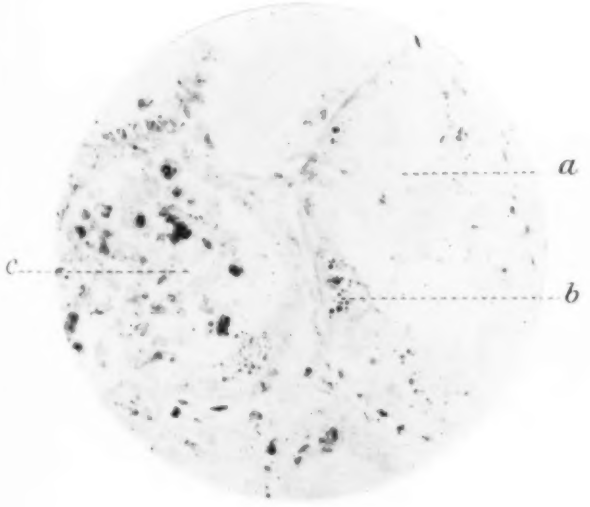

Fig. 5. Section of kidney showing the special fibrinous degeneration of the epithelium of the convoluted tubules. "A" indicates the granules in the degenerated cells staining by Weigert's method, and at "b" a cell almost entirely degenerated is seen. At "c" a number of degenerated epithelial cells in the lumen of a tubule can be seen, and many of these cells contain these granules.—*Dr. C. H. Potter.*

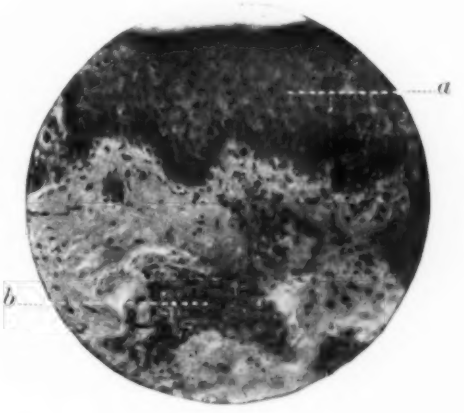

Fig. 7. Section of the skin showing the primary infiltration of the corium by the plasma cells. This is shown at "b", and "a" demonstrates that the epithelium is not yet injured.—*Dr. J. S. Fulton.*

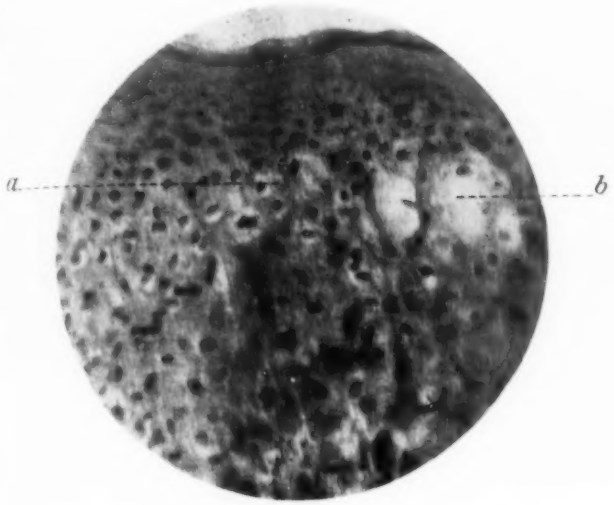

Fig. 8. Section through the epidermis in the papular stage. At "a" various stages of nuclear atrophy and fragmentation are seen, and at "b" the great edema and liquefaction of the cytoplasm of the cells can be made out. This is an example of the earliest epidermal changes in smallpox. The change in the cells at "b" is called the ballooning process, owing to the distended condition of the swollen cells.—*Dr. C. H. Potter.*

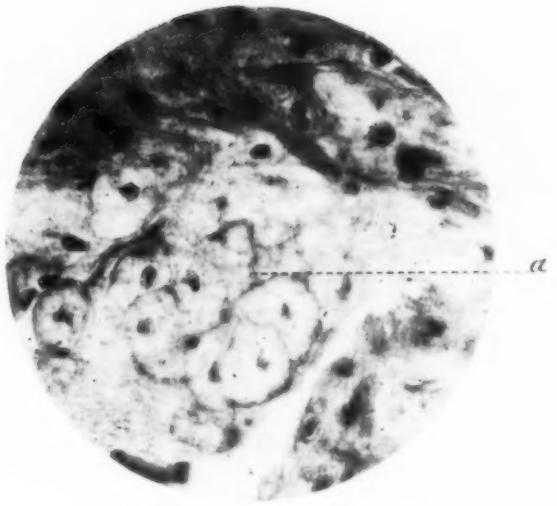

Fig. 10. Section through the epidermis showing a curious degeneration of the cells. The nuclei are shriveled, and the cytoplasm surrounding the nuclei is broken up into circular masses like fat drops. At "a," the phantom outline of these globular masses can be seen, while in the upper part of the picture the epithelium shows normal nuclei and cytoplasm.—*Dr. C. H. Potter.*

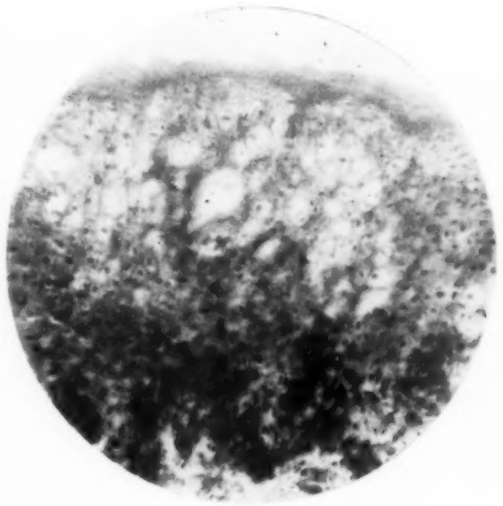

Fig. 9. Section through the epidermis. This shows a continuation of the process described in Figure 8. The various layers of the epidermis are shown in various stages of degeneration, and some of the cells are swollen to about three times their normal size, and the granular cytoplasm is replaced by a clear fluid. Some of the nuclei are present, but many of the epithelial cells have lost their nuclei. This is the stage which precedes the vesicular stage.—*Dr. C. H. Potter.*

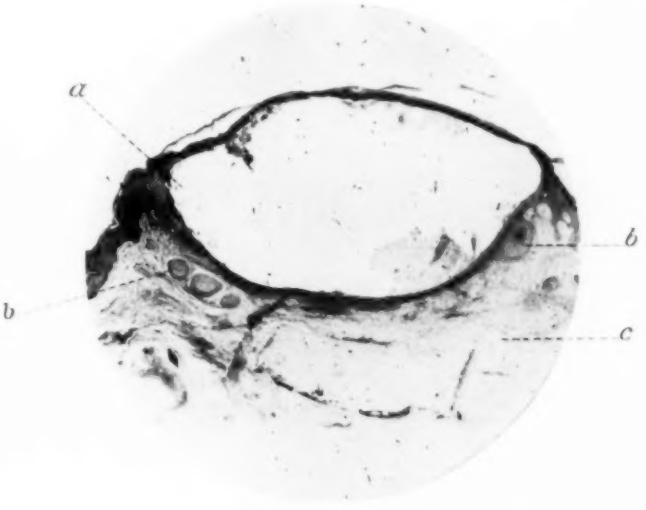

Fig. 11. Section through the skin showing a vesicle originally filled with a clear fluid. At "a," the junction of the stratum lucidum and the stratum granulosum, the layer of epidermis has split, and the layers pass above and below the fluid contents of the vesicle. This thin layer is unruptured, and the corium at "c" shows no infiltration. At "b" several hair follicles are seen.—*Dr. C. H. Potter.*

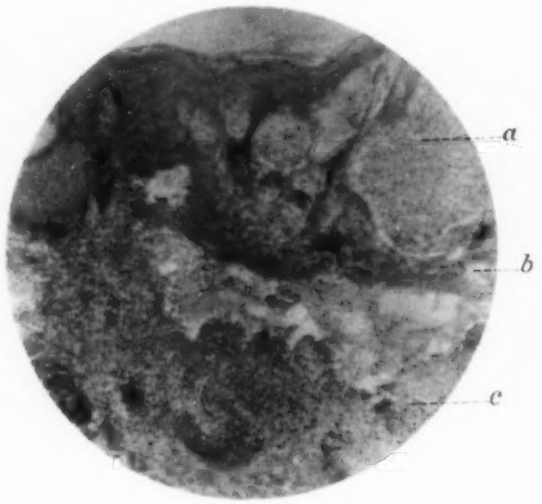

Fig. 12. Section of a pustule which has ruptured at "b," where the break in the rete Malpighii can be seen. "A" shows the pustular contents of the pustule, and "c" indicates the intense purulent infiltration of the corium. This explains the pitting, as in these cases the scar must always follow the inflammation of connective tissue, while the unruptured pustule will regenerate its epithelium without scar formation. The adjacent normal epithelium can be seen at the upper portion of the picture.—*Dr. C. H. Potter.*

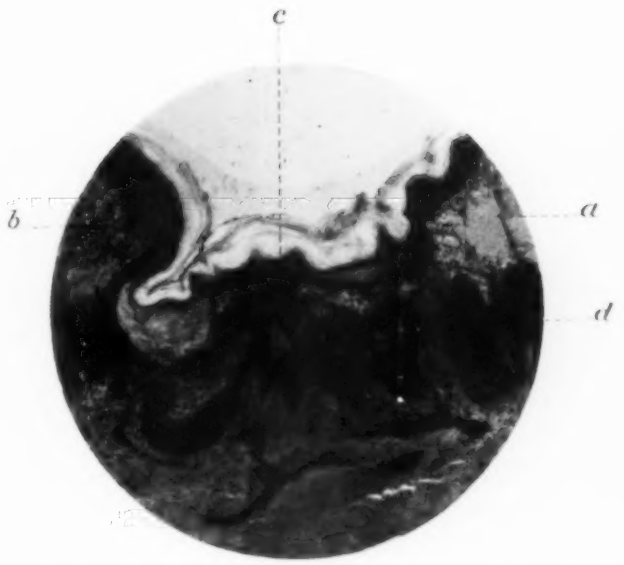

Fig. 13. Section through the central portion of a pustule showing the phenomenon of umbilication. The crater-like depression is seen at "c," and below this the various layers of the epidermis show very little swelling, edema or other changes. The edema and purulent infiltration to either side can be well seen. At "a" the layers of epidermis are separated by a sero-purulent fluid, and at "b" the exudation is entirely purulent. At "d" the lower layer of the epidermis is unruptured, and the corium shows no infiltration. This section shows the reason for umbilication very well.—*Dr. C. H. Potter.*

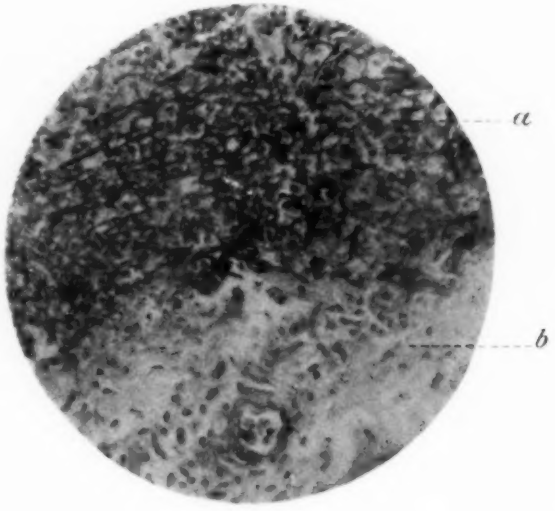

Fig. 14. Section at the base of a pustule just adjacent to the corium. This shows the fibrinous network, including pus cells often seen in pustules. At "a" this network is shown, and "b" shows a corium infiltrated by plasma cells. At the lower portion of the section a small vessel surrounded by plasma cells is seen.—*Dr. C. H. Potter.*

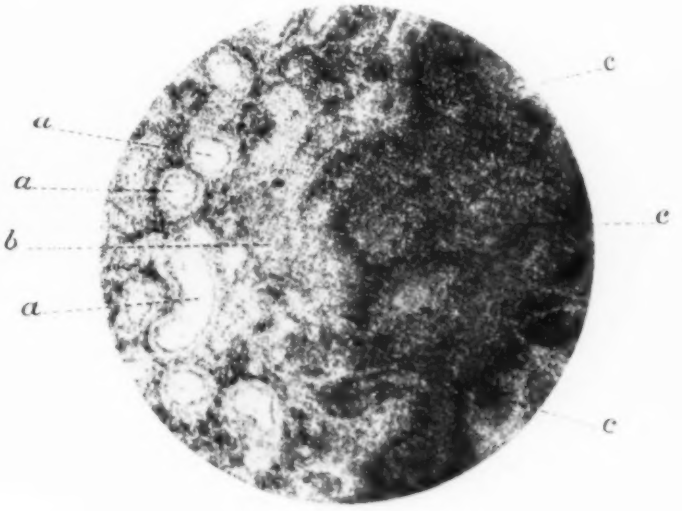

Fig. 15. Section of testicle showing the necrotic areas. "A" indicates the fairly normal seminiferous tubules, "b" the tubules undergoing necrosis and destruction, and "c" shows the completely necrotic zone with intense nuclear fragmentation.—*Dr. C. H. Potter.*

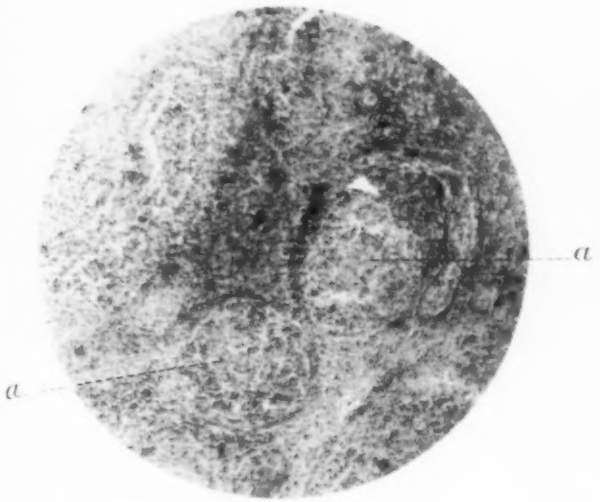

Fig. 16. Section of testicle under a high power, showing at "a" the remains of tubules which have undergone partial necrosis. Their outline as tubules can still be made out, but their gradual transition into necrotic material can be well seen.—*Dr. C. H. Potter.*

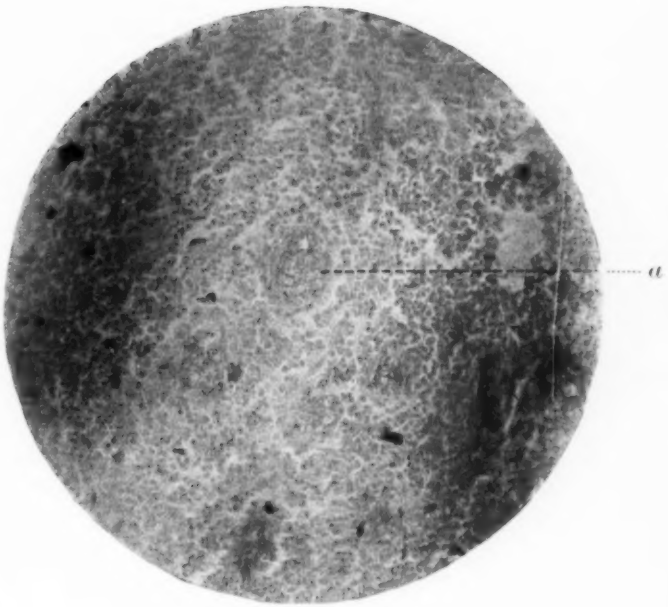

Fig. 18. Section of lymphatic gland showing at "a" a thrombus consisting of a fine reticulum of fibrin in a lymphatic vessel. The normal lymphocytes are well shown.—*Dr. J. S. Fulton.*

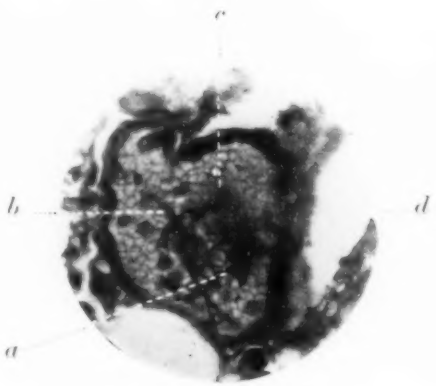

Fig. 17. Section of lung showing the beginning of thrombosis in a blood vessel. The strands of fibrin are well shown at "a," and "b" shows a few leucocytes. "C" shows a mass of red blood corpuscles, and "d" shows a clear air vesicle.—*Dr. J. S. Fulton.*

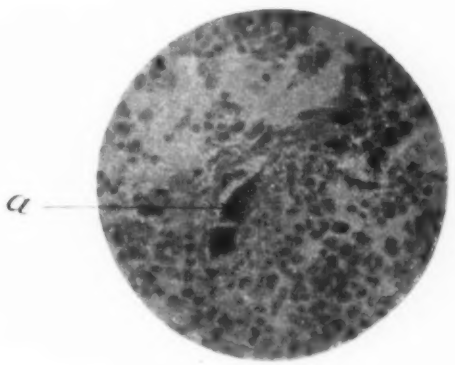

Fig. 21. Section of a lymphatic gland showing an embolus of micrococci in a small vein at "a."—*Dr. J. S. Fulton.*

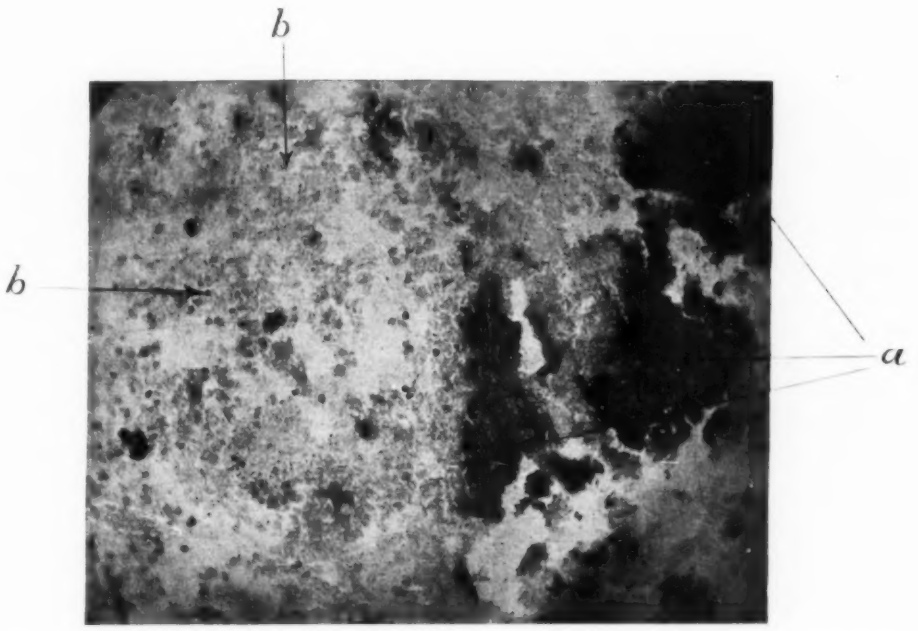

Fig. 19. Section of a mass of necrosis in the lung. The enormous masses of bacteria are well shown at "a," while "b" shows the necrotic material containing many necrotic cells.—*Dr. J. S. Fulton.*

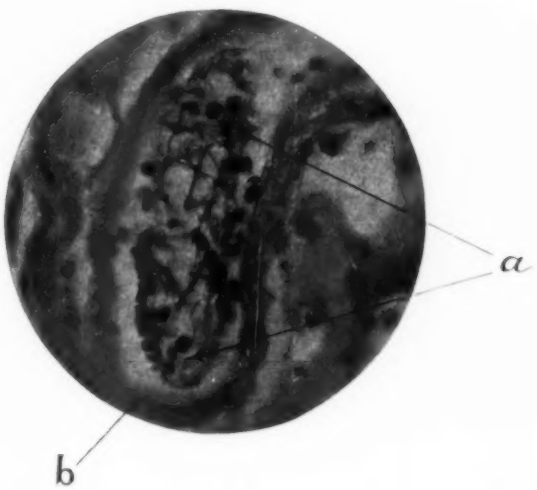

Fig. 20. Section of the testicle showing a vein containing a thrombus. At "a" many strands of fibrin are to be seen, while "b" indicates the wall of the vein.—*Dr. J. S. Fulton.*

From this point they are carried to the lymphatic glands by the lymph current, and stained sections from the cervical and bronchial glands gave a remarkable picture.

In the cortex of the glands, usually just beneath the capsule, there were extensive focal necroses. The lymph-sinuses were greatly distended, and the necrotic areas often contained neutrophilic leucocytes. When these glands are stained by Weigert's method, many of the necrotic areas are seen to consist almost entirely of masses of streptococci. There are also many streptococci in the lymph sinuses of the glands. The arteries and capillaries also contain streptococci, and there are fibrinous thrombi in many of the lymph channels of smaller glands. This condition was found in the cervical and bronchial glands, showing the absorption of the streptococci from the pustules of the skin, and the diseased mucous membrane of the bronchus.

It has been a disputed point as to whether the secondary infection with the streptococcus originates from the pustules of the skin or from the necrotic mucous membrane of the respiratory tract. It seems likely that both of these may be sources of infection.

In one of the cases, the mucous membrane of the epiglottis, larynx and trachea was the seat of a dirty gray membrane, which contained numerous streptococci in the necrotic surface layer when stained by Weigert's method. The lung itself was normal. The pustules on the skin contained a few diplococci, and the corium contained emboli of streptococci. This was a case of profound streptococcic infection in which large masses of organisms were found (see Fig. 21) in the cervical lymph glands, the kidneys, the liver, and the thymus gland. The tremendous masses of streptococci in the respiratory tract suggest this as the main atrium of infection. The organisms may also have entered the circulation from the skin, but they were so few in number as compared even to the viscera, that the skin may have simply been a secondary repository for the organisms brought originally from the respiratory tract.

In another case the streptococci undoubtedly came from the respiratory tract and the lung tissue. The trachea contained a membrane, the bronchi contained much pus, and there was a fibrinous pleurisy and broncho-pneumonia with many necrotic areas in the lungs (see necrotic areas in lung with organisms in Fig. 19). In all of these situations there were numerous streptococci, but neither the pustules on the skin nor the lymph spaces of the corium contained organisms. The viscera contained many streptococci.

In a third case the respiratory tract and lungs were normal, but the pustules on the skin, and the lymph spaces in the corium contained large groups of streptococci. This was a pronounced case of general infection with emboli of streptococci in the viscera, and these organisms undoubtedly entered the general circulation from the pustules.

These organisms then enter the general circulation, causing general septicemia, and bacteria were demonstrated in stained sections from the kidney, liver, lung, testicle, spleen and thymus gland. The bacteria were in the necrotic areas of the lung, the capillaries and the alveolar exudates, and also in the intertubular capillaries of the kidney, the lymph spaces of the portal system, the capillaries of the liver, the perivascular lymph spaces of the lung, the necrotic epithelium of the bronchi, and the veins and lymph spaces of the thymus gland, which also contained thrombi. The presence of the pyogenic bacteria in the skin and lymphatic glands has already been mentioned.

Ewing[6] found streptococci in the heart's blood in 29 cases of smallpox, and Arnaud[7] obtained this organism in two cases from the blood of the heart during life.

TENDENCY TOWARDS GENERAL THROMBOSIS.

Many of the viscera contain fibrinous thrombi in the small veins and lymphatics, and these lesions are usually very pronounced in the blood vessels of the lung, spleen, testicles, thymus glands and lymphatic glands. Large masses of fibrin are very frequent in the vessels of these viscera, and Fig. 17 shows the beginning of fibrin formation and thrombosis in a blood vessel of the lung. The strands of fibrin are well shown at "a," and at "b" lymphocytes and neutrophilic leucocytes are to be observed. "C" shows the mass of red blood corpuscles and "d" the open spaces of surrounding air cells. Fig. 18 is a photograph of a lymphatic gland, and at "a" the fine network of a fibrinous thrombus of a lymphatic vessel can be seen. Fig. 19 shows a collection of short chains of streptococci into masses, and represents a very frequent picture in smallpox sections. The large masses of organisms found in various viscera are often startling, and they might almost be described as emboli of microorganisms. The photomicrograph was made from an area of necrosis in the lung, and shows the micrococci collecting into masses resembling the agglutination of other organisms.

At "a" large groups of micrococci can be seen, and in the centre of the picture a number of individual micrococci can be distinguished. At "b" the various stages of coagulative necrosis with fragmentation of nuclei and granular destruction of the cytoplasm of cells resulting in homogeneous masses of necrotic material can be observed.

In many of the thrombi groups of streptococci can be stained, and these organisms probably injure the endothelium of the vessels, and thrombosis results. Fig. 120 shows a thrombus in a vein of the testicle, but no organisms were present in this section. The rather coarse fibrin filaments including red blood corpuscles in their mesh-work are indicated at "a" and the wall of the vein is seen at "b." "C" shows a few cells of the seminiferous tubules.

The liver is one of the favorite seats for the presence of streptococci in smallpox, and Fig. 3 shows a number of organisms in the capillaries of the liver between the rows of liver cells. The streptococci can be well seen at "a."

In some of the smaller capillaries of the testicle there are many large droplets which take up a fibrin stain. They almost resemble small hyaline casts.

SUMMARY.

Although we have only studied five cases of smallpox at autopsy, yet some conclusions can be drawn from the work.

The early skin lesions indicate that the primary infection in smallpox takes place in the lungs, probably by inhalation. The poison, when it enters the circulation, shows a selective influence on the epithelium of the skin and respiratory tract, and many cases are probably not further affected.

The serious and fatal lesions of smallpox are caused by the secondary infection from the skin and respiratory tract, and the infectious agent is usually the Streptococcus pyogenes. This organism is so distributed throughout the lesions as to explain most of the visceral changes, such as thrombosis, local necrosis, and the various pulmonary changes.

This streptococcic septicemia is the most striking feature of fatal smallpox, and if it were possible to overcome this condition by a special serum, the mortality from the disease would be greatly reduced.

In conclusion I desire to thank Dr. Caleb Rohrer for assistance in the autopsies, and to acknowledge the many courtesies received from Dr. S. O. Heiskell, Assistant Commissioner of Health at Quarantine.

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METHOD OF INSTRUCTION IN SURGICAL PATHOLOGY.

ABSTRACT OF PAPER READ BEFORE THE AMERICAN SURGICAL ASSOCIATION IN WASHINGTON, MAY 4, 1903.

BY J. C. BLOODGOOD, M. D., *Baltimore,*

Associate Professor of Surgery, Johns Hopkins University.

Clinical instruction in the out-patient department and in the hospital wards has and will always have certain defects and limitations as a complete method of instruction.

The question to be solved is: Can these limitations and defects be removed?

The method of instruction in the surgical pathological laboratory of the Johns Hopkins Hospital has reached a sufficiently satisfactory development to justify its presentation at least as a possible solution of the problem of teaching surgery.

Clinical instruction in the out-patient department and in the wards must to a very large extent be limited to the observation of the patients treated. The usual period of time spent by a student in the hospital dispensary and wards is two years. During this time many important surgical diseases may not be seen at all or so seldom that the student does not get a clear or lasting clinical picture of these rarer forms of disease. Of necessity examples of the various surgical diseases present themselves in no systematic order, and are so scattered that the student seldom, if ever, can observe in the dispensary or even in the ward at the same time the different varieties of affections of a single organ or part. Even in the ward it is very difficult to arrange that the student follow the patient from his admission through the operation and after-treatment and to present to him the diagnosis confirmed in the pathological laboratory with the result of the treatment when the patient leaves the hospital; the ultimate result many years afterwards can never be presented. Although the time between the admission and discharge of the patient is frequently an interval of a few weeks only, nevertheless it is a difficult problem to keep all the students of a group familiar with even the important details in the history of each patient.

These limitations and defects can be remedied to a very large degree by the method of instruction which has been adopted in the surgical pathological laboratory of the Johns Hopkins University.

We may describe this method briefly as follows:

The instruction given to the third year medical class in the surgical laboratory is divided into two parts. The first may be called the systematic instruction which is given by pamphlets, museum specimens, microscopic sections and illustrations. The second part may be called routine instruction, which is limited to the fresh material, sent to the laboratory from the operating or the autopsy rooms.

First Part.—For systematic work the third-year student at the beginning of the year comes to the laboratory one

afternoon each week. The class is divided into groups of not less than five and not more than ten. Each group is given a pamphlet and a box of microscopic slides stained and labeled. Near each group on a table, a rolling table if possible, are placed the museum specimens discussed in the special pamphlet given to this group. The pamphlet corresponds somewhat to a chapter in a text-book on surgery. It may have, for examples, titles like these: "Tumors and Inflammations of the Breast," "Malignant Tumors of Bone," "Inflammations and Tumors of the Thyroid," etc., and each pamphlet discusses the various affections of a special organ or part. In the first part of the pamphlet entitled "Tumors and Inflammations of the Breast," for example, the student is given the number of cases treated during the life of the clinic, in relation to the total number of surgical patients. This gives him at once an idea of the relative frequency of the diseases of this organ or part. Then follows a classification of the various diseases admitted for treatment in the surgical clinic. Here the student learns the relative frequency of the various affections of this organ. If no examples of certain rare affections have come to the clinic for observation, it is so stated. The pamphlet then gives a summary of the clinical history and picture, treatment, gross pathological appearance and microscopic study of the various diseases of the organ discussed in the pamphlet which have been observed in the clinic, and, most important of all, the ultimate result after leaving the hospital. This general summary corresponds somewhat to what is contained in a chapter of a text-book on surgery. It has the advantage, however, of a monograph, in that it gives the summary of a certain number of specific cases, in each of which the diagnosis has been confirmed by a pathological study and in which the ultimate result is known covering a period varying from a few months to many years. Descriptions of rare diseases not observed in the clinic can be made from the literature with illustration. The second part of the pamphlet gives the clinical history, the treatment, the description of the fresh specimen which is preserved in the museum, and which the student can find on the table near the group to which he belongs. Then follows the microscopic description, and the student will find in his box of slides a section taken from the specimen he has just examined.

The general part of the pamphlet the student can read at home in connection with his text-book on surgery. When he comes to the laboratory he is advised to take a museum specimen, selecting the various diseases of the organ in a certain natural sequence. This specimen has a surgical and pathological number which is indexed in the pamphlet, so that the student can quickly turn to the clinical history of this case, and as in the majority of instances some years have elapsed since the patient has left the hospital the pamphlet is able to give him the ultimate result. Each museum specimen therefore is one identified with an individual, whose clinical history and ultimate result of his disease the student can read, and he can examine the microscopic section of the museum specimen by which the diagnosis was confirmed. By a careful reading of the pamphlet the student is brought in contact with the relation between the clinical, the gross pathological and the confirming microscopic diagnosis. In addition in the pamphlet he will find photographs of the patient illustrating the clinical appearance, diagrams, photographs of the fresh specimen, X-ray photographs if made, and in some instances photographs of microscopic drawings, and quite frequently photographs of the ultimate result.

Each group, as stated before, is given a pamphlet and a box of microscopic slides, and near the group are the museum specimens discussed in the pamphlets. From time to time the groups exchange material, so that by the end of the third year a large field of surgery has been covered.

I believe this method presents surgery to the student in just as systematic a manner as a didactic lecture, with the great advantage that he reads instead of listens, and with his reading he has object lessons, illustrative of the text. I have found very little demonstration to be necessary. The student works quietly by himself, as he would in the dissecting room or chemical laboratory. He is brought in close contact with the disease, with each specimen of which he can get in a few minutes the entire history of the case and the ultimate result. This is impossible if he sees the patient only. While this work in the laboratory is going on, the third-year student observes patients in the out-patient department and in the surgical clinic. When, for example, he sees a patient with the clinical appearance of an exophthalmic goitre he will have read in his pamphlet a summary of the clinical history, treatment and ultimate results of all the cases of exophthalmic goitre treated in the hospital up to that time, he will have examined the museum specimens of the thyroids removed and the microscopic sections; he will have seen numerous photographs before and after operation. In the fourth year he will be much better prepared to observe his patients clinically in the wards; when, for example, he sees a case of exophthalmic goitre in the ward, he can go to the library in the hospital and in a few minutes review his third-year instruction on this disease. If during the third and fourth year it is his misfortune to see few or no examples of various important diseases, he will at least in his third year have read the clinical history, seen the photographs, examined the museum and microscopic sections of one or more examples. This systematic method therefore prepares the student to make better use of the patients he sees clinically in the dispensary and in the ward, and it fills the gaps which cannot be avoided in clinical teaching.

Although the course is called one of surgical pathology its ultimate aim is really clinical diagnosis, taught, however, from the museum specimen and microscopic section instead of the patient. Clinical instruction should and must always be considered the most important part of medical teaching, and this laboratory course is by no means a substitute, but a supplement, its only object being better to prepare the student for instruction in the dispensary or at the bedside.

Such a laboratory course has the great advantage that it utilizes the material accumulated during the entire life of

the surgical clinic. The student is brought in contact not only with the patients observed during his two years' course of instruction, but with the entire experience of the surgical clinic. This method stimulates better records, more careful descriptions of the fresh tissues removed at operation and autopsy, and their preservation in the museum. It becomes a necessity to keep track of the patients treated in the surgical clinic; one is stimulated to get better illustrations, the history of every important case, every museum specimen; microscopic sections and photographs are as accessible to the student as a book in a hospital library.

The preparation of the pamphlet is not specially difficult. Once made thay can be added to from year to year. The photographs necessary for a set of 5 to 10 pamphlets and the microscopic sections will last for years. The museum specimen is practically indestructible.

In addition to the systematic course just described, fresh material from operations and autopsies is used. In the first part of the year every fresh specimen is assigned at once to a third-year student. The probabilities are that he has seen this patient in the dispensary or in the surgical clinic; in any event he is instructed to make a summary of the clinical history and record the clinical diagnosis. He examines and makes a description of the fresh specimen and makes his own naked eye diagnosis. The afternoon on which the entire class meets in the laboratory those students who have been assigned fresh material meet the instructor in a small room. The cases are discussed and frozen sections are exhibited. After the first of January the class meets on two afternoons in the week. By this time the students have had the opportunity from the systematic course and from the study of a certain number of fresh specimens to acquire a certain amount of familiarity with surgical diseases, and for this reason the demonstration of the fresh material is made before the entire class. The cases are presented by the student to whom the specimen was assigned. By this time the student has been brought in contact with the various surgical diseases from different standpoints. He has studied a number systematically in the pamphlets, illustrated by the museum specimens and microscopic section. He has examined personally a number of fresh specimens in conjunction with the clinical history. He has seen a certain number of cases clinically in the surgical dispensary and clinic. For this reason the student can better appreciate now a general demonstration and discussion on the routine fresh material, so that while he is continuing to study by himself, the experience of the surgical clinic of the past, he is brought more and more in contact with the material of the present. It is my rule to exhibit those specimens which the student has seen as fresh material again when it has become an alcohol museum specimen, so that he can compare the difference between the fresh and the alcohol specimen. As much as possible the student is informed of the ultimate microscopic diagnosis.

About once a month a lantern slide demonstration is given; the first demonstration, for example, on Inflammation and Tumors of the Thyroid, would be given to the class, after this pamphlet had been studied by each group. I am quite positive that the time necessary to give such a course is no more than that which would be taken by a course of didactic lectures. A properly prepared pamphlet is certainly better than any notes of a lecture which can be taken by a student. He can digest most surely what he reads better than what he hears, especially if during his reading object lessons in the shape of photographs, museum specimens and microscopic sections are before him. The instructor can confine his talking to the demonstration of fresh specimens, to general remarks on certain more difficult problems, and to a certain amount of personal instruction. The method of teaching is not only more satisfactory to the student, but infinitely more so to the teacher.

Such a course may be a difficult one to establish, but when once established, it can be maintained with a minimum of labor and a maximum of results. The good results are not only to the student, but to the teacher and to the surgical clinic.

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NOTES ON NEW BOOKS.

The American Year Book for 1903. Medicine. Edited by GEORGE M. GOULD. (*W. B. Saunders & Company.*)

In noticing the annual edition of this work one can only repeat the favorable opinions of previous years. It is turned to so frequently and has become so familiar that perhaps with use we are apt to forget how helpful it is. Not only the last volume but the previous ones are often employed for reference. Dr. Gould is to be congratulated on the continued ever excellence of the work.

The Medical Annual. A Year Book of Treatment and Practitioner's Index. (*Bristol: John Wright & Co. New York. E. B. Treat & Co., 1903.*)

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